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The Concept of the Fatal Dose

Death on the Sigmoid

The current difficulties and disagreements about what constitutes the fatal dose of a poison are well brought out by quotations from two internationally recognized and authoritative authors, who (although they are dealing specifically with arsenic) illustrate a fairly general toxicological problem. Glaister,[1] e.g. states: "The smallest recorded fatal dose (of arsenic) is 2 grains." Smith,[2] on the other hand, writes: "It is generally stated that 2 grains (0.13 gramme) is a fatal dose of arsenic, but there appears to be little or no authority for such a general statement. In my experience, recovery after much larger doses is common, and I have not seen a death from a dose as small as 2 grains."

There is a fairly widespread belief that every poison has a fatal dose which can be relied on to kill, but this is a standpoint which needs considerable qualification.

In practice as well as in experiment it is seldom a simple matter to define the lethal concentration of a drug precisely, and the attempt is often made to meet the medico-legal problem in a different way (equally controversial and equally fallacious) by quoting the least amount known to kill (i.e. the minimum lethal dose or MLD). The unsatisfactory state of affairs is revealed by contradictory and vague generalizations, often based on what are really inadequate case reports.

The genuine paucity of this type of information in Man is partly due to the fact that the data must necessarily be derived from the investigation

1. Glaister, J. L. (1950): *Medical Jurisprudence and Toxicology*, 9th ed., p. 547. Edinburgh: E. & S. Livingstone Ltd.
2. Smith, S. (1949): *Forensic Medicine*, 9th ed., p. 456. London: J. & A. Churchill Ltd.

of discovered crime or accident. There is clearly, therefore, ample cause for lack of clarity in this field of inquiry. The obscurities are, however, further increased by a failure to appreciate the nature of the biological problem involved. Age, sex, weight, nutrition, etc., are all factors known to have a potent influence on the effects of drugs and these variable factors contribute much to an understanding of the apparent vagaries encountered in the study of the behaviour of poisonous substances in Man.

To investigate even one clear-cut, all-or-none end-point such as that provided by the fatal dose of a poison requires rigid control of the variable factors. Such a procedure is an elementary requirement recognized and observed as a routine in all methods of biological standardization. A closer look at the way these matters are conducted under laboratory conditions may throw much light on the matter and illustrate an important biological principle.

The strength of digitalis, e.g., is measured by its effects on various animals. One of the most satisfactory tests for this purpose is based on the capacity of digitalis to kill frogs. The death of the animal is an unequivocal end-point which can be observed in sufficiently large numbers of frogs to produce statistically reliable results. In the frog test for the potency of digitalis preparations, animals in the different groups are sexed, weighed, starved for the same period before the experiment, etc. and then distributed throughout the test as wholly comparable groups. The digitalis preparations under test are adjusted for constant volume of each injection and the size of the dose is related to the body weight of each animal.

The results of such an experiment, carried out under rigidly controlled conditions, show that the lethal effect of the drug is not directly proportional to the increase in the size of the dose administered, as commonsense may lead one to expect. The fatal results, when plotted graphically against the dose given, demonstrate that the relations between the dose administered and the effect observed follow an unusual pattern, thus bringing to light a novel biological relationship between the two events under investigation. The results form an S-shaped (or sigmoid) curve of the general type illustrated in Fig. 1.

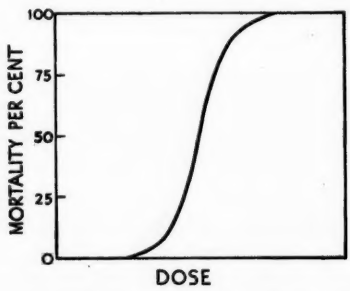


Fig. 1. A sigmoid (S-shaped) curve characteristic of many biological dose-response relationships.

In the first place, there is a range within which no animals are killed. In the second place, although all the animals have been chosen most carefully as similar in all respects, a particular dose kills only a definite proportion of the animals injected, until very large doses are reached; even then, in

some experiments, it may be found that this result is not always consistent. The curve (Fig. 1) is seen to rise most steeply in the region of the dose that is lethal to 50% of the animals injected (the dose known as LD 50) and provides one of the convenient and reliable points for assay purposes. This is a zone in which dose and response are more nearly directly proportional.

Another interesting feature disclosed by animal experiments is the behaviour of the survivors. If these frogs are rested, fed and used subsequently for other assays, animals which lived through previous experiments will succumb in the later tests.

Experimental data also show how misleading attempts may be to measure a minimum lethal dose. Burn[3], reviewing the fundamental contributions made by Trevan, demonstrates very forcefully the fallacies inherent in an attempt to express the minimum lethal dose, unless there is an appreciation of the over-all statistical context in which the matter must be viewed. He writes:

'. . . the attempts usually made to determine a minimum lethal dose are exposed to great inaccuracy. Let us consider for a moment a definition of a minimum lethal dose as that dose which kills 2 out of 3 rats. If there are 99 rats in a large cage and every one of the rats is injected with this dose, two-thirds will die and one-third will survive. But let us take them from the cage 3 at a time. Is it possible to be sure that 2 will die and 1 will survive out of the first 3 we inject? Of course, there is no certainty. There are 33 rats in the cage which will survive the injection, and it is easily possible that 3 of these may be chosen. Only in a certain proportion of groups taken out of the cage will 2 rats die and 1 survive. In other groups, death will occur in 3 out of 3, or in 1 out of 3, or in 0 out of 3.'

All these facts have an interesting bearing on the problem in Man. Individual lethal effects cannot be predicted with any certainty at all; the lethal probabilities for a particular dose can be calculated (if adequate data are available) and a non-fatal dose on one occasion may be lethal to the same animal on another occasion. Moreover, as the relation of the dose to its response is not a directly proportional one, the true relationship can only be appreciated by a statistically satisfactory analysis which, in animal populations, will fit either an S-shaped or some other characteristic type of curve.

It is against this background that contemporary contradictions in standard textbooks must be viewed. Indeed, this approach resolves the difficulties and demonstrates that what appear to be conflicting opinions are in fact wholly compatible views. When the statistical nature of the issue is realized, the problem involved in a single particular case is the better appreciated. For the individual, death lurks somewhere along the sigmoid.

3. Burn, J. H. (1937): *Biological Standardization*, pp. 10—11. London: Oxford University Press.

Legal Relations of the Mentally Ill

A Functional Approach*

Elwyn L. Cady, Jr.†
University of Kansas City School of Law, Kansas City, U.S.A.

A recent article in a popular magazine[1] is headed in large red type: 'Who's Right—The Lawyer or the Doctor?' The article itself is responsibly written and does not amount to a diatribe that so often appears against law and lawyers by scientists or the reverse attack on scientists by lawyers or judges. It does, however, publicly focus a feeling that there exists a basic antagonism in Forensic Psychiatry and Psychology.

Much professional literature does reflect rather violent feelings among men trained to think in radically different patterns.[2] Many refuse to consider the probability that there has been too much 'right or wrong' talk in this arena already. When men incompletely understand each other, it is often found that tempers flare. To counteract this trend, the 'law-science movement'[3] has sprung forth with the idea that what is needed is an affirmative integration of the efforts of persons trained in the law and of those trained in science. It is this spirit which has prompted this *Journal* and others like it.

The fundamental tenet of the law-science approach can be stated simply: In an age of science, those human principles designed to regulate human conduct ('rules of law') should be firmly grounded upon insights developed by the various sciences.

To achieve this goal, there must be a fundamental semantic co-operation of lawyer and scientist. 'Communication barriers' have been erected between professional men as their individual specialties have grown. As each area of

*This article is also currently appearing in the Ohio State Law Journal Symposium Issue on *Law and Psychology*.
†Director, Law-Medicine Programme, University of Kansas City School of Law.

[1] Deutsch, *Woman's Home Companion*, June 1952, p. 44. Similar titles have recently appeared in the technical literature. For example, see Shindell (1953): *Medicine vs. Law: A Proposal for Settlement*, J. Amer. Med. Assoc., **151,** 1078.
[2] The main currents of this 'conflict talk' are set out by Hall, *Mental Disease and Criminal Responsibility*, Col. L. Rev. **45,** 677 (1945), substantially reprinted (1947) as Chapter 14 of *General Principles of Criminal Law*, 477.
[3] Smith (1953): *The Philosophical and Practical Basis for the Synthesis of Law and Science*, Texas L. Rev. **31**, 625.
Editorial (1951): *The Tulane University Law-Science Program*, Tulane L. Rev., **25,** 214.
Brosman (1949): Editorial: *Two Recent Program Additions* (1949): Tulane L. Rev., **24,** 108.

investigation has progressed, shorthand expressions ('technical jargon') have appeared. Communication is thereby facilitated among members of the specialist group, but a verbal 'iron curtain' is drawn as far as the specialist in another discipline is concerned.[4]

It has been the lawyer's task traditionally to serve the function of 'humanist' as regards technical information. In a case where some scientific fact or theory is in issue, the advocate has been faced with a problem of digesting available technical knowledge and then translating it into readily understood terms for benefit of a tribunal lacking in specialized education or knowledge. Because of this history, scientists rightly expect lawyers to lead the way in forging law-science linkages. It will be our purpose here to call attention to several landmark efforts in this evolution in what has been called 'psychological law.'[5]

I. Contracts and Wills

In civil cases the lawyer often encounters problems of mental illness in contract cases and will contests. Other instances in which such problems arise in a similar vein include guardianship proceedings, litigation involving family problems and tort actions against the mentally ill. We will confine ourselves to contracts and wills in this discussion.[6]

Perhaps the most illuminating study in this field is that fashioned by Green on mental incompetency.[7-11] Reaffirming the view that the term 'insanity'

[4] This professional isolationism has been well recognized by forward-looking psychiatrists. 'Law and medicine, because they have each been pursuing their own ends independently, have come to talk, as it were, different languages. The aims of both professions are, however, I believe, the same, so far at least as they affect the common weal, but they have thus far been unable to get together largely because they do not understand each other. Each profession has its background and traditions and habits of thought radically different from the other and finally each in his turn comes to talk its own language, which members of the other profession do not understand. There is a sore need for overcoming this difficulty of understanding, but so long as doctor and lawyer meet only as witness and cross-examiner there is little hope. We must get together and work together, and it is on the basis of the recognition of a common purpose that an understanding will come.' White, *The Need for Co-operation Between the Legal Profession and the Psychiatrist in Dealing with the Crime Problem*, A.B.A. Rep. **52**, 497, 498 (1927).

[5] Wharton speaks of the specialty of psychological law in his treatise on *Mental Unsoundness, Embracing a General View of Psychological Law* (published as Book 1, Wharton and Stille's *Medical Jurisprudence* [3d ed., 1873]).

[6] A more all-inclusive presentation is that of Guttmacher and Weihofen (1952): *Mental Incompetency*, Minn. L. Rev., **36,** 179, which forms the substance of Chapter 14 of their treatise, *Psychiatry and the Law*, 323 (1952).

[7] Green (1940): *Public Policies Underlying the Law of Mental Incompetency*, Mich. L. Rev., **38,** 1189.

[8] Green (1941): *Judicial Tests of Mental Competency*, Mo. L. Rev., **6,** 141.

[9] Green (1943): *Fraud, Undue Influence and Mental Incompetency: A Study in Related Concepts* Col. L. Rev., **43,** 176.

[10] Green (1943): *The Operative Effect of Mental Incompetency on Agreements and Wills*, Texas L. Rev., **21,** 554.

[11] Green (1944): *Proof of Mental Incompetency and the Unexpressed Major Premise*, Yale L. J., **53,** 271.

has come to have little usefulness because of its semantic abuse, he substitutes 'mental incompetency', 'that type or degree of mental disorder, in any particular case, which is legally significant and which produces a different legal result (from what) would have flowed from the same situation had not that particular type or degree of mental disorder been present.'[12]

As I interpret Green's work, the following propositions seem to have great force:

1. Public policy underlying this law consists of derivations of 2 fundamental interests:

(*a*) *Security:*

1. '(I)t is the policy of the law to protect the security of transactions;
2. '(I)t is the policy of the law to protect the security of acquisitions; and
3. '(I)t is the policy of the law to protect the social institution of the family.'[13]

(*b*) *Equality:*

'People are not equals in any real or substantial sense, and the law has recognized this truth. In realizing the glaring discrepancy between the real and the ideal, the law has sought to remedy the situation by producing equality where inequality had heretofore existed. It has developed a policy of equalization, which it has put into effect by extending a measure of protection to those classes of persons who are not, in any real sense, the equals of their contemporaries in the society in which they live.'[14]

This protective policy assumes 3 forms:

1. 'Protection of the public or of society at large from the acts of the mental incompetent.
2. 'Protection of the incompetent from society, because of the unequal position which he occupies toward his fellow men.
3. 'Protection, not of society, nor of the incompetent himself, but of the family or dependants of the incompetent.'[15]

2. In establishing judicial tests for mental incompetency, i.e. drawing a line of demarcation between the 'normal' and the 'mentally incompetent,' a subjective standard is ostensibly applied whereas in reality an objective standard is adhered to. Though the traditional 'understanding test'[16] is stated as the rule of law, judges of necessity cannot probe the inner reaches of the mind. Rather, '(t)hey base their judgments, just as the psychiatrists do, upon the behaviour of the person investigated, only they do not admit they are doing it. Having examined (from the evidence produced at trial) the behaviour of the individual, insofar as that behaviour was revealed to the court, the judge (or jury) then passes upon the question as to whether or not that behaviour conformed to the standard set by the law. Since the standard is phrased in terms of mind, and not in terms of behaviour, the fact finder draws the necessary inference of the state of the individual's mind from his observed behaviour. In other words, he must translate his actual finding into the language of the standard which has been set up. But the fact remains that what is actually happening is that judges, no less than

[12] Green, *supra*, note 7, at 1191-92.
[13] Green, *supra*, note 7, at 1207.
[14] Green, *supra*, note 7, at 1207-208.
[15] Green, *supra*, note 7, at 1213.
[16] *In re* Humphrey, 236 N.C. 141, 71 S.E. 2d 915, 917 (1952).

psychiatrists, are employing an essentially behaviouristic technique. The difference lies in the fact that the judges do not do it deliberately, but still conduct their inquiry as if they were not examining behaviour, but instead were scrutinizing mind.'[17]

3. Since the law is necessarily concerned with separating somewhat arbitrarily those persons who are 'normal' and those who do not meet the test of 'mental competency,' cases which fall close to the line often present difficulties. In order to compensate for lack of a 'sliding scale of legal effects,' the law is able to mitigate possible harsh results in these borderline cases by integrating concepts of fraud and undue influence with those of mental incompetency.[9]

4. In establishing the operative effect of a finding of mental incapacity on wills or contracts (i.e. are they 'void' or 'voidable'), courts must weigh 2 conflicting public policies. 'One policy is to protect the incompetent or his dependants. The other is to uphold the security of transactions. These policies are at war with each other. If full recognition is given to the former the instrument will be held utterly void. If full recognition is given to the latter the instrument will be held valid. However, the modern tendency is to adopt neither of these extreme positions but rather to effect some sort of compromise between these conflicting policies. The compromise is effected under the guise of calling the instrument 'voidable,' but in measuring the legal consequence flowing from this concept the courts try to work out a result which will balance the competing equities in the concrete case.'[18]

5. Evidentiary facts used to establish the probandum of mental incompetency can be conveniently classed in 4 groups:

(*a*) '(S)ymptomatic conduct of the alleged incompetent;
(*b*) '(O)pinion testimony of incompetency;
(*c*) '(O)rganic condition and habits of the alleged incompetent;
(*d*) '(M)oral aspects of the transaction and its consequences.'[19]

Green submits that probative value increases as one goes down the list. Indeed, he suggests that a sub-category of (*d*) is often controlling. This consideration is 'whether or not the transaction conforms to the normal pattern of similar transactions.'[20] The touchstone of this entire thread of the law as concerns the Science of Proof,[21] is forcefully distilled by Green with an assist from Llewellyn:

'It is submitted that in determining the issue of mental incompetency, more frequently than otherwise, courts are passing upon the abnormality of the transaction rather than on the ability of the alleged incompetent to understand the transaction. To rephrase this thought in terms of legal doctrine, we might say that since, both in unconscious desire and in articulate effort, the court is seeking evidence on whether mental incompetency *has affected the particular transaction*, the dominant factor in the evidence is whether

[17] Green, *supra*, note 8, at 161. See also Malan, *The Behavioristic Basis of the Science of Law*, A.B.A.J. **8**, 737 (1922), A.B.A.J. **9**, 43 (1923).
[18] Green, *supra*, note 10, at 589.
[19] Green, *supra*, note 11, at 275.
[20] Green, *supra*, note 11, at 298-99.
[21] *Vide infra*, note 38.

the court sees *the particular transaction in its result* as that which a reasonably competent man might have made.'[22]

The reader is, of course, referred to Green's masterful analysis of the cases in this field which found the general propositions set out. It has been the pitfall of many lawyers, and more particularly of scientists, to be unable or unwilling to look beyond the bare statements of rules of law for their functional underpinnings. When the more pervasive principles are examined, however, I feel that increased understanding is mutually promoted between scientists and lawyers. It amounts to a process not unlike depth-analysis of the psychiatrist, the meticulous dissection of the anatomist, or the astute probing for cause and effect of the medical clinician.

II. Hospitalization of the Mentally Ill

Current attention as regards hospitalization is largely centered on the *Draft Act Governing Hospitalization of the Mentally Ill*.[23] Since hospitalization of the mentally ill is now predominantly within the scope of State medicine, it was entirely proper that an agency of the Federal Government concern itself with drafting this important model legislation. It can be hoped that within a relatively short span of years, the several States will see fit to consider and adopt the Act in its essential features.

Again, we see the older term 'insanity' discarded in favour of more useful terminology. By Section 1, (*a*), a mentally ill individual is defined as '(a)n individual having a psychiatric or other disease which substantially impairs his mental health.'[24] Here we are more properly dealing with a clinical diagnosis of psychiatric illness than we were when considering mental incompetency. Goals of the law are different in the 2 situations. The rationale of hospitalization has to do with treatment and rehabilitation. With mental incompetency, we were more concerned with the ability of the individual to carry on certain transactions. There is a valid reason, therefore, for establishing clinical criteria of mental illness when hospitalization is at issue, though we feel justified in looking to a standard of 'fairness or unfairness of the individual transaction' in litigation involving mental incompetency.

Under provisions for voluntary hospitalization, treatability appears as the primary concern. Any person who is mentally ill under the definition cited, or who has symptoms of mental illness, comes within the purview of this part of the Draft Act. Of course, effective implementation of such provisions in hospitalization laws is beyond present contemplation due to the shortage of facilities and trained personnel—a reflection of traditional public apathy toward the mentally ill.

[22] Green, *supra*, note 11, at 307-308.

[23] Public Health Service Publication No. 51, *A Draft Act Governing Hospitalization of the Mentally Ill* (1952). The text of the Act, devoid of commentary, is reprinted in Davidson (1952), *Forensic Psychiatry*, 377.

[24] Draft Act, *supra*, note 23, at 4.

With involuntary hospitalization the criteria for admission involve not only the fact of mental illness, but also additional social factors. Under the standard nonjudicial procedure with certification by 2 designated examiners, it is required 'that they have examined the individual and that they are of the opinion that:

(*a*) He is mentally ill; and

(*b*) Because of his illness, is likely to injure himself or others if allowed to remain at liberty; or

(*c*) He is in need of treatment in a mental hospital, and because of his illness, lacks sufficient insight or capacity to make responsible application therefor.'[25]

It becomes apparent, then, that there is no necessary correlation between 'mental incompetency' and 'mental illness which is treatable or which may endanger the physical safety of the patient or others.' In the past, these terms have too often been confused as a result of using the term 'insanity.' To-day we find persons who require hospitalization but who are not mentally incompetent. So also, there are mentally incompetent individuals who could not benefit from hospitalization and who are not patently dangerous to others. Justice is certainly not fostered by lumping all of these people together under the label 'insane.'

A scope note prefacing the Draft Act indicates that the definition of mentally ill person excludes the mentally defective, but is intended to 'include persons who are psychopaths, chronic alcoholics, or members of other special groups . . . to the extent that the individual meets the criteria set forth in the definition.'[26] This brings us to a consideration of one of the greatest enigmas confronting law and medicine to-day—what to do about the so-called 'psychopath' or 'neurotic character'—the person who is not 'out of touch with reality' but whose disorder lies in the fact that he suffers from 'a form of neurotic reaction which has as its principal symptom a deviation of behaviour which is antisocial and undesirable by present day moral and legal standards.'[27] In short, his disturbance in personal adjustment is 'taken out on his environment' rather than internalized as is the case with other neurotics.

Traditionally, the state mental hospitals have been primarily devoted to the care of psychotic individuals. Often, when 'psychopaths' were admitted upon request of law enforcement officers or others because of violent behaviour, these people caused such disturbances in the hospital that administrators were wont to release them as soon as possible. Since many psychiatrists adhered to a 'gentlemen's agreement' type of approach to 'sanity hearings', they were willing to equate 'insanity' with 'psychosis', and since the 'psychopath' was not technically psychotic, they opined that he was not 'insane' and so was eligible for release despite his potential for destruction.

[25] *Id.* at 6.
[26] *Id.* at 1.
[27] Strecker, Ebaugh and Ewalt (1951), *Practical Clinical Psychiatry*, 311, 7th ed.

Since the newer legislation contemplated under the model Draft Act makes provision for hospitalizing 'psychopaths,' it will be up to the public to demand facilities for their treatment (if treatable) or their social isolation (if they are sources of danger to the community).

These are only the more apparent offspring of the clarification in definition of mental illness. Others are certain to follow if the legislation is widely adopted. Our thought in stressing a revised terminology has been to contrast 'mental incompetency' with 'mental illness' (as defined in the Draft Act). Now let us turn to another semantic problem involving 'responsibility' and 'accountability.'

III. Criminal Responsibility or Accountability

The professional literature abounds in discussions of mental states and the criminal law. My limited purpose here is to invite attention to 3 crucial constellations of inquiry in this field.

1. Substitution of 'accountability' for 'responsibility' in the legal test of mental disease sufficient to establish a defence to crime;
2. Substitution of 'clinical language' for 'punitive terms' in the rehabilitation of criminal offenders;
3. Basic changes in criminal law administration to uphold the law and yet salvage human resources now being dissipated.

The first of these hinges on a very provocative article that appeared recently.[28] Instead of inquiring whether 'the party accused was labouring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing; or, if he did know it, that he did not know he was doing what was wrong,' the inquiry becomes, 'was the defendant suffering from disease of the mind and if so, was it sufficient to render him unaccountable under the law for the crime charged?'[29] As explained by the authors, '(t)he question then becomes not whether his knowledge of the quality or nature of the act, and his comprehension that he was doing wrong was affected, but whether the total personality (i.e. the ego), was impaired by mental disease to a degree rendering him unable to adjust to society's rules.[30] . . . The anticipated benefit in expert testimony by the use of the concept "accountability" is in the direction of providing an objective description and analysis of subjective phenomena in persons accused of crime in conjunction with an estimation of the degree of ego impairment resulting from his illness. In estimating the impairment of the whole organism, not merely a traditionally restricted area of mental function will be the subject of the test, but the degree of disturbance of total personality

[28] Bromberg and Cleckley (1952): *The Medico-Legal Dilemma: A Suggested Solution*, J. Crim. L., Criminology, Police Sci., **42**, 729.

[29] The older language is quoted from M'Naghten's case, 10 Clark & Finney 200, 210; 8 Eng. Rep. 718, 722 (1843). The suggested test is that of Bromberg and Cleckley, *supra*, note 28, at 742.

[30] Bromberg and Cleckley, *supra*, note 28, at 744.

function will be examined . . . What the psychiatrist estimates is the degree of socio-biologic impairment (illness) of the person in terms of present-day psychological knowledge. The jury would decide to accept or reject the psychiatrist's opinion of how accountable the prisoner is without recourse to the "knowledge of right or wrong" test. The suggested test is broader and more realistic. It is not applied to an arbitrary isolated "moral sense" but to the functional capacity of the actual human organism as he is encountered in practical life and in medical study.'[31]

This approach to the problem of determining criminal accountability is certainly of the functional variety, but many are concerned lest we centre all our attention on 'insanity as a defense in criminal cases.' Is not the post-conviction stage worthy of more emphasis? Since juries can be capricious and 'convict the innocent' or convict the mentally ill who are nevertheless 'accountable' in their eyes, what of these victims? What of the many convicts who manifest social or emotional pathology that does not enter into the 'defense of insanity'?

Again, in this, our second inquiry, new concepts are being applied. Recently, the Attorney General of the United States announced[32] that the Federal Youth Corrections Act[33] would be promptly implemented.[34] Clinical terms of 'treatment and rehabilitation' replace 'punishment and retribution.' Rumblings of the many ways in which modern dynamic psychology and psychiatry are being brought to bear in this movement are cogently spotlighted by a distinguished lawyer and psychoanalyst, Marcel Frym.[35] When the insights of scientists are effectively marshalled actually to treat and rehabilitate criminal offenders (the process now under way) we can look forward to a further step which may mitigate problems inherent in the first 2 inquiries.

This third improvement, though perhaps appearing radical at first blush, has actually been advocated for many years by persons learned in the law. Wharton's analysis, published 80 years ago, to my mind is a classic and deserves quotation:

> If the views taken in the preceding sections be sound; if in the first place, there are inherent difficulties in the way of making insanity a ground of defense on the trial of a man who, on this hypothesis, is psychologically incapable of either tendering or preparing any such issue; if, in the second place, we must recognize sanity and insanity as pressing into each other gradually at a line that cannot be judicially defined, each being capable of various degrees; if, in the third place, it be right that the present system of confinement of insane criminals be remodelled—then it will become necessary for those to whom

[31] *Id.* at 744-45.
[32] K. C. Times, 23 May 1953, p. 1, col. 3.
[33] 64 Stat. 1085 (1950), 18 U.S.C. Ch. 402 (Supp. 1951).
[34] See also Bennett, *Blueprinting the New Youth Corrections Program*, 15 Federal Probation, September 1951, p. 3.
[35] *The Criminal Intent: Outdated Concept*, Texas L. Rev., **31,** 260 (1953). In addition, see Hacker and Frym (1949): *The Legal Concept of Insanity and the Treatment of Criminal Impulses*, Calif. L. Rev., **37,** 575. In earlier years, Karl Menninger spearheaded this development. Menninger (1945): *The Human Mind*, 443-60, 3d ed.; *Medicolegal Proposals of the American Psychiatric Association*, A.B.A. Rep. **52**, 486 (1927); *The Psychiatrist in Relation to Crime*, A.B.A. Rep. **51**, 751 (1926).

the work of legislation is committed to amend the law so as to require the question of insanity to be determined by a competent tribunal *after* a conviction of the fact of guilt. For the following undeniable evils result from the present system:

(*a*) A tribunal of at least but secondary competence is charged with the determination of the most difficult and yet most momentous question to which human observation can be applied.

(*b*) A subject is introduced into the question of guilt or innocence, as to which no fixed judicial rules can be laid down, and which really concerns only the character and the extent of punishment.

(*c*) We find by this process the *sane* convict; the *malignant insane* convict, who requires discipline and is in some degree morally responsible; the *innocent insane* convict; and the *lunatic*, who is in confinement but is not charged with crime: for all of whom there is in some jurisdictions but *one* common method of discipline provided, viz. that of the penitentiary; in others, but *two*, that of the penitentiary and of the ordinary lunatic asylum. The result of this is acquittals in some cases, where there should be convictions; convictions in other cases where should be acquittals, and in almost all cases an erroneous system of punishment.

The remedy for these difficulties is one to which we must come sooner or later, and for which the common law has been from the beginning always striving, and yet losing from almost its very grasp. It is to confine the inquiry before the court and jury to the mere *factum* of the commission of the offence; reserving the question of the treatment to be determined by a special commission of experts, to be appointed for the purpose of examining convicts alleged to be insane. The proposition to be put by the court to the jury, in such circumstances, is not: 'Was the defendant capable of judging between right and wrong?' (a proposition which no jury can determine) but: 'Did he', as a matter of fact, 'commit the specific act charged?' For whether he committed it as sane or insane, the result is, if the offence in point of law is indictable, that the safety of society requires that he should be placed in seclusion for such a period as will promote the joint ends of personal reformation and the preservation of the well-being of the community at large. If he be guilty without the palliation of mental infirmity, certainly the severest penal code—with the single qualification of cases of murder in the first degree—can ask nothing more than this. If, on the other hand, he was at the time labouring under mental derangement, in no other way can the extent of his responsibility be accurately determined and the proper degree of discipline adjusted. For this great question of sanity or insanity can really only be determined by those to whose daily and hourly care the convict is committed, and who have thus full opportunity of inquiring into his antecedent as well as his present condition.

'Thus,' to adopt the language of an intelligent commentator, 'except as regards the curative course to be adopted, on our view of the case, the subtle line of distinction which there have been so many abortive attempts to draw, between criminal and non-criminal lunatics, is of no practical importance, and the unavailing search, unless as a matter of metaphysical speculation, may be abandoned as unnecessary. In either case, the person concerned, whether called a lunatic, or an ordinary criminal, should be so placed as to put it out of his power to inflict further injury, and to afford the most likely means for his cure.' And thus, also, not only will the sanction of human life and property be protected from the recurrence of those monstrous acquittals, by which, under the plea of insanity, the most dangerous criminals are suffered to run at large, but the interests of humanity will be subserved by a proper discipline, as well as a just classification, of those whose accountability is diminished or destroyed.[36]

[36] Wharton, *op cit. supra*, note 5, at 764-69. In conjunction with the citation of nineteenth-century commentators, a remark by Val Satterfield, Chairman, Psychiatry Section, American Academy of Forensic Sciences, is instructive. He calls attention to the tendency of modern students of forensic psychiatry to gloss over the writings of nineteenth-century observers and in so doing to lose a number of potent insights. (Personal communication.)

By thus abolishing, in effect, the defense of insanity, a fundamental change in the philosophy of criminal law would be effected. It is evident that society is not yet ready for such drastic reform.[37] A pre-requisite, it seems, will be a successfully established rehabilitation programme that has vigour if the lurking constitutional objections are to be overcome.

In working through the functional interrelations of these 3 facets of criminal justice, we gain a clearer realization of the distinct problems that arise in the criminal law as opposed to the psychiatric problems found in civil litigation. Let us now move into the arena of the court room and look at witnesses and parties that may be mentally ill.

IV. The Mentally Ill on the Witness Stand and as Parties in Personal Injury Litigation

The ramifications of this subject have not yet been explored in any great detail. A broad framework for investigation, however, has been constructed by Wigmore and Smith. Wigmore lists 'mental derangement' as one of the generic human traits to be considered in analysing testimonial evidence and cites a copious literature, much of which, however, is outdated.[38] We need a modern series of systematic psychological studies of witnesses, it seems to me, before we can crystallize functional generalizations that amount to more than 'court room hunches,' valuable as these may be.[39]

Smith has been developing a series of comprehensive investigations involving problems of mental illness as they arise in personal injury litigation.[40]

[37] Judge Parker, in a majority opinion of the Supreme Court of Washington, considers the State's argument that modern humane treatment of convicts 'practically removes them from the realm of punishment and places them in a position but little different from those other unfortunate members of society which the State is obliged to care for and restrain of their liberty; not because they have committed wrong, but because of their menace to society and themselves without fault of their own, the insane . . . The argument seems to be, in its last analysis, that because of modern humane methods in caring for and treating those convicted of crime, there is no longer any reason for taking into consideration the element of will on the part of those who commit prohibited acts, when their guilt is being determined for the purpose of putting them in the criminal class for restraint and treatment. Learned counsel's premise suggests a noble conception, and may give promise of a condition of things towards which the humanitarian spirit of the age is tending; yet the stern and awful fact still remains, and is patent to all men, that the status and condition, in the eyes of the world and under the law, of one convicted of crime is vastly different from that of one simply adjudged insane. We cannot shut our eyes to the fact that the element of punishment is still in our criminal laws.' State *v.* Strasburg, 60 Wash. 106, 110, pp. 1020, 1025 (1910). See also Tulin (1932): *The Problem of Mental Disorder in Crime: A Survey*, Col. L. Rev., **32,** 933, 952-53; Rood (1910): *Statutory Abolition of the Defense of Insanity in Criminal Cases*, Mich. L. Rev., **9,** 126.

[38] *The Science of Judicial Proof*, 345, 3d ed., 1937.

[39] Burtt (1931): *Legal Psychology* manages only a single paragraph on this topic (p. 115). Recently, however, more study has been given this fertile area. See, e.g. note, *Psychiatric Evaluation of the Mentally Abnormal Witness*, Yale L. J., **59,** 1324 (1950), and Orenstein (1951): *Examination of the Complaining Witness in a Criminal Court*, Amer. J. Psychiat., **107,** 684.

[40] *Cross-examination of Neuropsychiatric Testimony in Personal Injury Cases*, Vand. L. Rev., **4,** 1 (1950); *Psychic Interest in Continuation of One's Own Life: Legal Recognition and Protection*, U. of Pa. L. Rev., **98,** 781 (1950); Smith and Cobb (1944): *Relation of Emotions to Injury and Disease: Legal Liability for Psychic Stimuli*, Va. L. Rev., **30,** 193; Ann. Int. Med., **19,** 873 (1943); Smith and Solomon (1943): *Traumatic Neuroses in Court*, Va. L. Rev., **30,** 87; (1944): Ann. Int. Med., **21,** 367.

Through the active co-operation of many scientists in diverse fields, he has been extremely successful in weaving together a functional approach to the so-called 'traumatic neuroses and psychoses.' Since the law-science integration in this field is undergoing rapid development currently, it is rather premature at this juncture to attempt detailed report on its project. Suffice to say we are presently hammering out the fundamentals in the court room (through medico-legal consultation work), in the class room (through lectures, demonstrations and discussions in forensic neurology and psychiatry), in special lectures (to claims men and plaintiff's attorneys in particular), and in medico-legal research projects emphasizing intensive student participation.

General Conclusions

From our survey of several problem areas in the legal relations of the mentally ill, it is possible to suggest certain guides for the future development of forensic psychiatry and psychology.

1. Broadly conceived notions of law-science integration are required.[41] No longer can the scientist shrug his shoulders and say that these medico-legal problems do not concern him. Lawyers and judges cannot continue to dodge their responsibility to act in the light of the best psychological information extant.

2. Communication barriers must be bridged between scientific specialists and lawyers. Until men in these various disciplines make deliberate efforts to become conversant with the various view-points that have been developed in the 'other man's province', future progress is problematical. The limited number of men to-day trained in both science and the law cannot 'go it alone'. They require active co-operation from all sides.

3. Public apathy toward problems of the mentally ill has been reflected in the archaic condition of statutory law in many States. For the most part, provision for periodic revision to keep pace with current situations is non-existent. Lawyers have a special duty in this field[42] and many are showing increased interest in the problems involved.[43]

4. In this review, I have examined the view-points of several authorities who have made significant contributions toward our understanding of basic problems in limited areas of psychological law. It is not represented that this amounts to a thorough survey or an extensive analysis. The aim

[41] 'It is not easy to over-estimate or to exaggerate the importance of the subject;—the great difficulties that surround it,—its bearing upon personal liberty and life,—the rights of property and the safety of society,—all enhance the necessity of approaching insanity with a true appreciation of the strength of the ramparts behind which it is intrenched.' Elwell (1881): *A Medico-Legal Treatise on Malpractice, Medical Evidence and Insanity*, 371-72, 4th ed.

[42] Melaney (1950): *Commitment of the Mentally Ill: Treatment or Travesty?* U. of Pitt. L. Rev., **12,** 52.

[43] The American Bar Association has had a Special Committee on Rights of the Mentally Ill for 10 years, A. Ba. A. Rep., **68,** 183 (1943).

has been merely to throw a spotlight on several of the leading guideposts to the future.

5. Finally, we must recognize the tentative nature of our present insights and give heed to the counsel of a great legal scholar, Bishop:

> This subject of insanity is practically difficult. Men of sane mind know themselves but imperfectly, and they comprehend others less than themselves; nor is there language to convey, in exact form even the little knowledge we possess of the sane mind. When, therefore, we undertake to investigate the phenomena of insanity, to discuss them, and to deduce from the principles of law the legal rules to govern them, we are embarrassed with difficulties which should make us cautious, and restrain us from any extensive laying down of doctrines for unseen future cases. So that *caution* should guide judges, counsel, and juries in their investigations of insanity. They may well restrict their theories to the particular facts in issue, and though they accept the aid of experts it should not be overlooked that they are liable to err. . . . . We think ourselves wiser upon this subject than were our fathers; undoubtedly we are; but there is wisdom yet to be acquired.[44]

Background student research has been provided by Mr. Warren Donaldson of the Forensic Neurology and Psychiatry class, and by Mrs. Barbara Logan who is studying in the Department of Psychology.

[44] I. Bishop (1892): *The Criminal Law*, 240, 8th ed.

Medico-Legal Mythology

Some Popular Forensic Fallacies*

H. A. Shapiro, B.A., Ph.D., M.B., Ch.B., F.R.S.S.Af.
Cape Town

1. The Alleged Value of Microscopy in the Diagnosis of Live and Still Birth.
2. Some Aspects of Age Determination.
3. The Time of Death:
   (*a*) The Development of Rigor Mortis.
   (*b*) Allegations About the Rate of Cooling of the Dead Body.
4. Specific Tests for Human Blood.
5. Does Arsenical Poisoning Preserve the Dead Body?
6. The Misuse of the Term 'Cyanosis'.
7. The Liquidity of the Blood in Asphyxia.
8. Some Conclusions About Vulgar Errors.

1. The Alleged Value of Microscopy in the Diagnosis of Live and Still Birth

In South Africa Section 306 (1) of Act 31 of 1917 reads:

'On the trial of a person charged with murder or culpable homicide of a newly born child, such child shall be deemed to have been born alive if it is proved to have breathed, whether or not it has had an independent circulation, and it shall not be necessary to prove that such child was, at the time of its death, entirely separated from the body of its mother.'

Although the emphasis is on breathing as the crucial test, no statutory criteria for establishing that the child has breathed are laid down. We therefore have to depend on the current body of scientific knowledge and opinion to come to a conclusion on the matter.

Apart from an inspection of the organs *in situ*, 2 tests are usually described. The first and most important, applicable only in the absence of putrefaction of the lungs, is the hydrostatic or flotation test; the second requires a microscopic examination of the lungs.

Eminent obstetric and forensic authorities maintain that there are very clear-cut microscopic differences between the lungs of infants born dead (i.e. who have never breathed) and of newly born infants who breathed for a short while before dying. These differences, we are led to believe, can settle the issue of live or still birth beyond doubt.

*Based on an address delivered at a meeting of the Medico-Legal Society (Johannesburg) on 27 February 1953.

In 1945 Smith[1] stated:

'It is advisable to cut microscopic sections of the lung if there is any doubt about the occurrence or extent of respiration. If the child has breathed, the lung tissues will show expanded alveoli (air sacs) which readily differentiate such a lung from one which has never respired.'

He has since modified his views (in the 9th edition of his book) in the light of the facts to be described here, and first published in *Clinical Proceedings*[2] in 1947. His illustrations, however, remain as before.

For the purpose of this discussion we confine ourselves to the microscopic criteria in the lungs for the diagnosis of live and still birth, i.e. we will not inquire into the floating test.

The literature abounds in dogmatic and unambiguous descriptions of the microscopic appearances under discussion. One eminent obstetrician (Gibberd)[3] states that before birth the future lung air spaces (or air sacs referred to by Smith) are completely collapsed and there is not any space in any part of the lungs. Theoretically this seems not unreasonable.

The post-natal human lung may be compared crudely with a bunch of grapes in which the main stem and its various branches correspond to the windpipe and its subdivisions, and the grape skins themselves (if they are imagined to be empty) correspond to the ballooned-out little air sacs, microscopic in size, in which the subdivisions of the air passages (the ultimate twigs of the bunch of grapes) terminate. According to Gibberd, therefore, in the unborn child before delivery the walls of the grape skins are completely in apposition.

Many of the microscopic descriptions have been supplemented authoritatively and convincingly by photomicrographs.

It is generally written and taught that the wall of the air sac, i.e. the grape skin, is lined (in the unborn child shortly before it is born) by tall rectangular cells. As soon as the infant is born, with its first breath and cry, it inflates the air sacs. The resulting distention is supposed mechanically to flatten the rectangular cells lining the wall of the air sac so that the post-natal appearance of the human lung is acquired.

Glaister[4] and Smith[1] are among many who have implied that there is no difficulty in distinguishing microscopically between lung tissue of a newly born infant which has not breathed and that which had participated in extra-uterine breathing. They have also published illustrations to support their contentions.

Neither Glaister nor Smith indicates the approximate age of the infants or foetuses from which their specimens are taken. It will be seen later that this is an important omission.

It takes very little practical experience of foetal lung microscopy to convince the investigator that there are considerable difficulties in distinguishing the lungs of the stillborn from those who have died recently after delivery, even if the latter have breathed. The textbook criteria were

found to be sufficiently obscure and in conflict with experience to suggest that this matter be put to the test. On the principle that the proper study of mankind is man (and not rats), I did this by investigating a series of 30 cases, with intra-uterine ages ranging from 6 weeks to full term. Wherever possible the history was checked, the intra-uterine age was determined, the post-natal age was obtained (from relatives or from the midwife) and the hydrostatic (floating) test was performed on the lungs (to see whether they contained air, and had therefore breathed). Putrid cases were excluded from the investigation. Microscopic investigations were made from known cases of live and of still birth, particular attention being paid to cases at or near full term. An attempt was then made to correlate the results of the floating test with the microscopic appearances, on the basis of the criteria generally recommended.

It became obvious immediately that this was a far from simple task, especially when the microscopic sections from cases at or near full term were mixed and examined in haphazard sequence without knowledge of the case histories. The percentage of error was very high in the hands of experienced medico-legal pathologists to whom the microscope slides were shown. The conclusion was that the current microscopic criteria were unacceptable because they were misleading. The difficulties have arisen from a failure to appreciate the embryological changes which normally occur in the course of the development of the respiratory tract in the human foetus.

When the human foetal test material was analysed, it was appreciated that some sections of lung tissue were encountered almost identical in appearance with those of the cases depicted by Glaister and by Smith; but these invariably came from relatively early embryos of about 4 months' intra-uterine age. On the other hand, in many cases of stillbirth at or near full term, the lungs of the child which had not breathed showed definite evidence of space formation in the air sacs, although no single part of these lungs floated in water.

Careful study of the whole series suggested a new hypothesis to explain the appearances observed. In the course of the development of the foetus there is a gradually evolving change in the structure of the respiratory tract, consisting of the well-known progressive branching of subsidiary tubes from the main windpipe in the early embryo. In the still ante-natal stages this process actually goes on to the formation of air sacs containing well-defined spaces with fairly thin walls (i.e. grape skins). This growth and development occurs in the absence of extra-uterine breathing (Figs. 1-8).

Such a gradual development of the lungs up to the moment of birth makes reasonably intelligible the manner in which extra-uterine breathing can begin with immediate success the moment the infant is born. If at birth the lungs, just before the first breath is taken, are an almost solid organ (as is maintained by the orthodox exponents of medico-legal tests),

the structural changes which would have to take place with a single breath seem to be quite incomprehensible both from the anatomical and the physiological points of view. The cases depicted by Glaister and by Smith

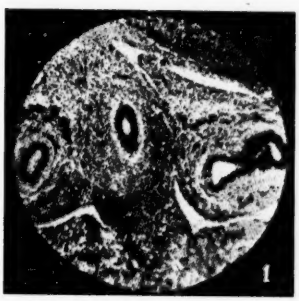


Fig. 1. Section taken from a five weeks' human foetus, showing early branching of the developing endodermal tree.

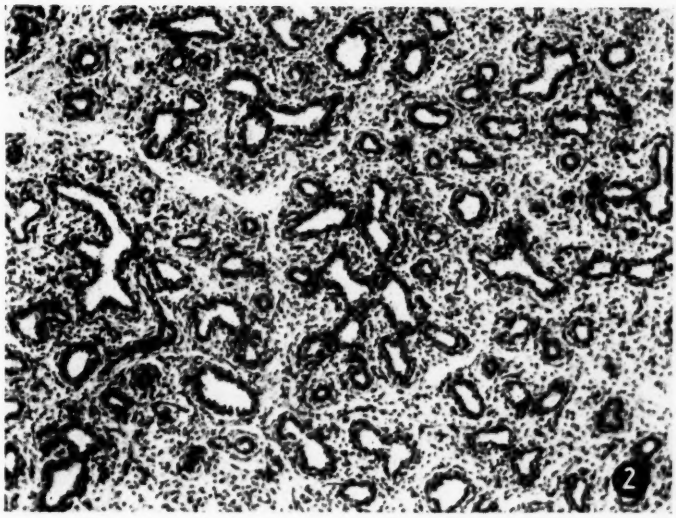


Fig. 2. Section from the lung of a foetus between four and five months of age. The lungs of this foetus did not float in water. Note the glandular appearance of the organ with the considerable amount of connective tissue between the ducts which are lined by cuboidal-columnar cells. Crown-rump length, 15 cm.

are accurately described as the lungs of foetuses of about 4-5 months' intra-uterine age. They are not characteristic of the lung appearance at or near term, when the medico-legal problem arises.

The omission by such authorities as Smith and Glaister to state the

age of the specimen from which their microscopic sections were taken now becomes exposed as a serious matter. The sections they have illustrated are actually of greater value in suggesting the age of the foetus than whether it has breathed. Indeed, the question of respiration is an academic issue at such an early embryonic stage.

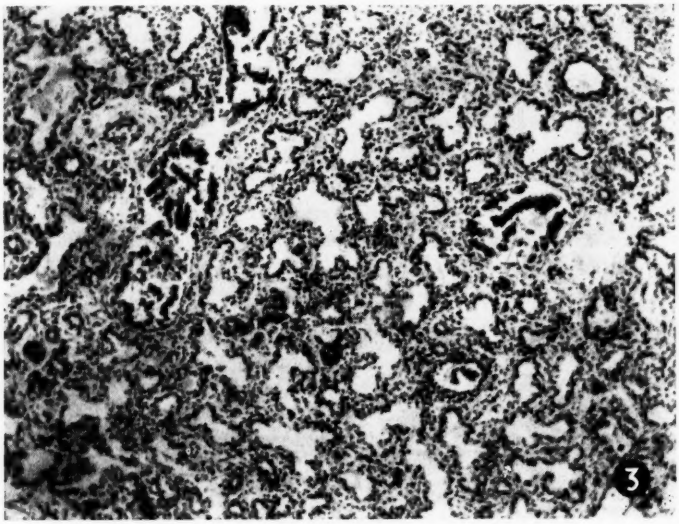


Fig. 3. Section from a five months' foetus. The lungs as a whole did not float in water, and this section was taken from a small portion which also did not float. There is evidence of a greater degree of subsidiary branching of the ducts, leading to the formation of spaces lined by cells which are less columnar than those seen in Fig. 2. Crown-rump length, 19 cm. Centre in calcaneum present.

The evidence indicates that, for purely embryological reasons, the lungs have reached such a stage of development just before delivery that their microscopic appearances will not differ appreciably from those of the lungs of newly born infants who have lived and breathed, if only for a short time, and whose lungs float in water. It is only because the lungs normally develop to the stage of air sacs before the infant is born that it is, in fact, in a position to breathe and survive immediately after delivery.

In cases of still birth, therefore, microscopic examination of the lungs may not only be unhelpful, but unless the concept of an evolving air sac pattern, independent of extra-uterine breathing, is kept in mind, microscopy may actually be misleading and suggest an entirely erroneous diagnosis of extra-uterine respiration and, therefore, of live birth.

This view of the gradual development of the lung pattern can be tested experimentally on animals. Ham and Baldwin[5] have examined a series of

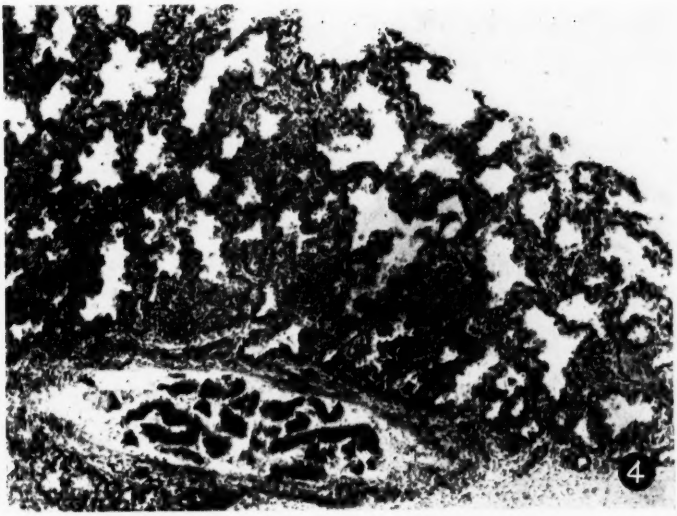


Fig. 4. Section from a six to seven months' foetus, whose lungs did not float in whole or in part. The subsidiary branching has proceeded to such an extent as to give the appearance of a fairly well opened up alveolar pattern. The interalveolar tissue is very cellular, leading to the appearance of thick-walled alveolar spaces.

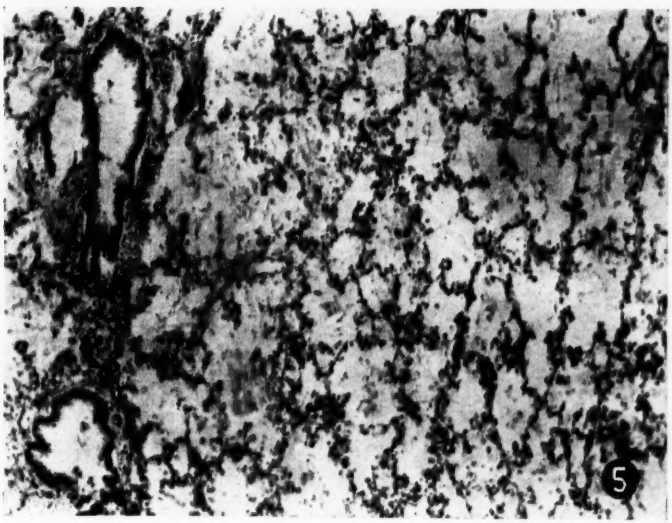


Fig. 5. Section from a still-born foetus at full term. No portion of the lungs floated in water. The alveolar pattern appears almost fully developed.

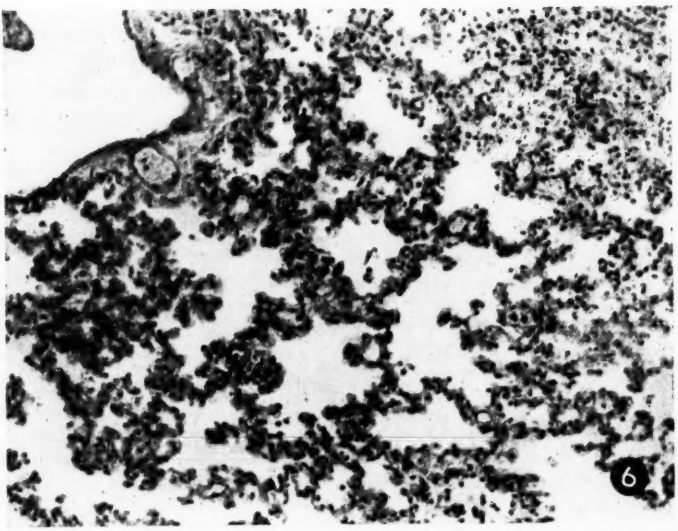


Fig. 6. Section from the lungs of a full-term foetus found lying free in the abdominal cavity in a case of death due to a ruptured uterus following an impacted transverse lie. No portion of the lungs floated in water.

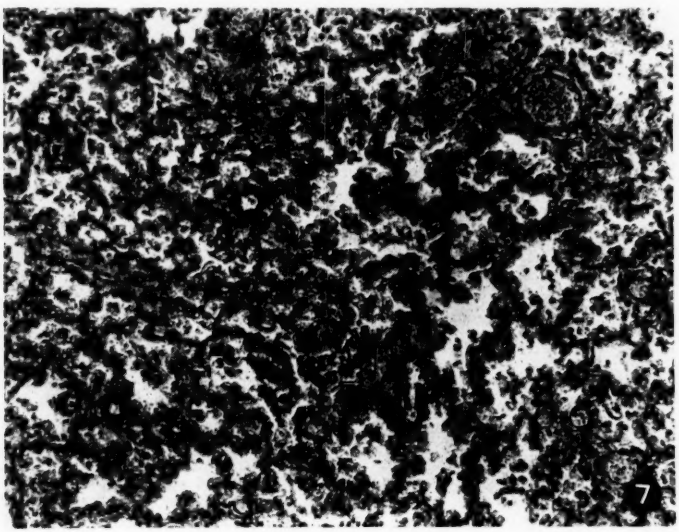


Fig. 7. Section from another portion of the lungs in the case referred to in the previous figure. In addition to obvious alveolar spaces in some areas, other portions of the lung have not yet opened up appreciably.

foetal lungs from pigs whose gestation periods were rigidly controlled. Their results confirm and strikingly corroborate in all respects the hypothesis advanced on the basis of the human material examined. Microscopy, therefore, appears to have a very limited role in deciding the medico-legal problem of live birth.

If the solid type of lung is found, then this is good evidence that the lung comes from a fairly early embryo. In such a case, it is unlikely that the question of breathing will seriously arise. In fact, on this basis, it can confidently be predicted that the examples of unrespired foetal lung illustrated by Glaister in the 9th edition of his book on p. 403, Fig. 197, and

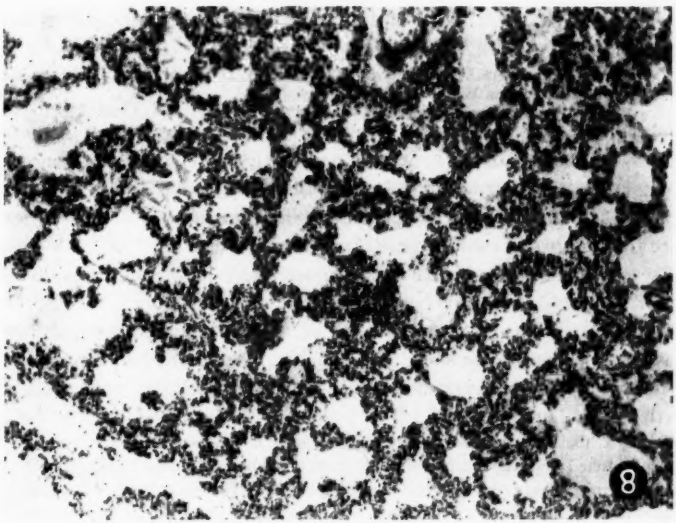


Fig. 8. Section from a full-term foetus which died a few minutes after birth. Death was due to a tentorial tear with intracranial haemorrhage. It is not possible to distinguish this section taken from a portion of the lung which floated, from those shown in Figs. 5 and 6, which were taken from cases of still birth in which no portion of the lungs floated.

by Smith (9th edition, p. 350, Fig. 152B) are not from cases at or near full term. If, on the other hand, a foetus is at or near full term, or has lived only for a few minutes, microscopy cannot always provide clear and unequivocal evidence of extra-uterine breathing. More often than not the scientific evidence will only permit the statement that the lungs came from a foetus at or near term. The microscopic appearance *may* be less equivocal after respiration has been well established at full term for a considerable period of time; but the kind of evidence illustrated in current standard textbooks on forensic medicine is actually only of assistance in throwing light on foetal age rather than on live birth.

These observations raise interesting speculations about the quality of expert testimony. On the face of it, a pathologist who relies on the current criteria advocated, particularly when he supports his opinion with the statement that he has made a microscopic study of the lungs, would seem to have made out an unanswerable case. What he has, in fact, demonstrated is that he has not been handicapped by a knowledge of the subject.

2. Some Aspects of Age Determination

In terms of the Births, Marriages and Deaths Registration Act No. 17 of 1923, a viable child is defined as an infant after 6 months of intra-uterine existence*. This is, therefore, an important age for accurate determination. Keith Simpson[6] states:

'The age of the foetus may be fixed with near mathematical accuracy by direct measurement and the appearance of the ossification centres.'

(Most bones develop from cartilage, i.e. gristle. An ossification centre is that place in the cartilage where the process of ossification, i.e. bone formation, begins.)

In common with many other authors he gives a table of these measurements correlating chronological age, body length and body weight and enunciates the empirical rule of thumb that 'the length in inches equals twice the number of months gestation (after four months)'.

The ossification centres are well characterized, constant in position and appear at fairly well-defined times, biologically speaking.

While certain data on the foetus can be obtained by measuring its length or its weight, the ossification centres must be examined by dissection in the dead body or radiologically in the quick or the dead. However, Rhodes, Gordon and Turner[7] state:

'Owing to the variation in the length and weight of newly born infants, these factors cannot be relied upon in determining the state of maturity.' (Some authors claim that the crown-rump length measurement is more reliable.) They also state: 'An examination for the ossification centres in the bones of the foot and the lower end of the femur is the most reliable method of determining the state of maturity of the newly born infant.'

Keith Simpson[8], however, states:

'The centres of ossification show such wide variations in time of appearance and rate of growth that they are of doubtful value in estimating the period of gestation.'

The ossification centre in the heel bone is usually given as being present at the end of the 5th intra-uterine month; that in the ankle bone at the end of the 7th month and that at the lower end of the thigh bone at the end of the 9th month, i.e. at full term.

It is a common enough experience to apply to a foetus a battery of the 3 tests of length, weight and ossification centres and to find that the results are not concordant. This makes the determination of age a highly arbitrary act, especially when single bones and their ossification centres are relied on. The anatomist or the radiologist with an extensive survey of all the bones may make a better guess. While some experts seem agreed that

* This refers to the Union of South Africa.

the greatest value is to be placed upon an examination of the ossification centres, it is extremely doubtful whether the bald statements in contemporary forensic textbooks are an adequate guide to the solution of this problem.

Students of skeletal maturation have long recognized that important factors influence the rate of appearance of ossification centres. One of the most important and most neglected has been emphasized by Pryor[9], who states:

'The bones of the female ossify in advance of those of the male. This advance may be measured in the early stages by days, later by months, and subsequently by years. . . . The bones of a first child ossify, as a rule, sooner than those of subsequent children.'

His conclusions were based on the study of 994 radiographs of which 140 were of foetuses between 10½ and 38 weeks; 100 newly born infants from 1 hour to 7 days old; 554 children between 3 months and 12 years and 200 from 12-23 years old.

Flecker[10] who regards Pryor's work as 'of monumental importance', endorses Pryor's views including the statement that 'in the ossification of the human skeleton, the female is in advance of the male from the earliest developments of centres of ossification in the embryo and throughout intra-uterine life, progressing from days to weeks and months'.

The medico-legal significance of the sex differences in the appearance of the ossification centres is apparently not generally known or appreciated. This may account for the neglect by forensic pathologists of the implications of these facts which explain why, if radiological examinations are made of unborn children about 4 weeks before term, a well-developed ossification centre will be found present at the lower end of the femur in some unborn infants but not in others. This apparent discrepancy is entirely explained by the sex of the child, the centre appearing much earlier in the females. There can, therefore, be an error of as much as 3 to 4 weeks in relying upon the ossification centre at the lower end of the thigh bone, if no attention is paid to sex, as is usually the case in medico-legal textbooks. Despite Pryor's suggestive work, it is not known precisely what the quantitative sex differences for the centres in the thigh bone, the heel bone and the ankle bone are. We do not know the limits of variation in the time of appearance of the centres for each sex separately, nor do we know to what extent the curves for the appearance of the centres in the 2 sexes overlap. We can be quite certain that there is such an overlap.

If, therefore, weight and length are not very reliable guides and if there can be an error of almost a month when reliance is placed upon ossification centres without taking the sex of the infant into account, the problem of determining the particular intra-uterine age of 6 months (of such importance medico-legally) becomes obvious and considerable, if not incapable of precise solution. A quantitative survey, taking into account race, sex, nutrition, etc., is clearly indicated as an important piece of forensic research.

An interesting application of this knowledge is to the determination of sex before birth. Radiography of pregnant women is a commonplace routine ante-natal procedure near term to study the relationship of the child to the mother. Inspection of the films, if the expected date of delivery is known, at about 4 weeks before term, should put the radiologist in a position to predict fairly accurately the sex of the unborn child. At my suggestion Dr. A. A. Meyer and Dr. J. N. Jacobson of Cape Town undertook such a survey on some 30 pregnant women. They were correct in their predictions of the sex of the unborn infants in at least 20 of these cases, including one of twins. This is probably significantly better than can be explained by guessing.

It is doubtful whether Pryor's claim can be substantiated that he could sex skeletons on the basis of the ossification centres present. Thus, apart from satisfying a pardonable parental curiosity, it is difficult to see to what other use this knowledge can be put at present.

In royal and Mohammedan circles (where the matter of a male issue is regarded as important) the medical practitioner may be in a position to comfort and sustain the hopeful.

Table I: The Appearance of Carpal Bones for Boys and Girls expressed in Percentage Present†

Bone	*Sex*	*Age*																	*Probable Age of Appearance in Months*
		B	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	
Capitatum	F	8	96	100															6
	M	2	98	100															6
Hamatum	F	8	96	100															6
	M	2	98	100															6
Radial*	F	0	78	100															9
Epiphysis	M	0	34	98	100														15
Triquetrum	F	0	20	52	79	100													23
	M	0	22	50	57	92	84	93	100										24
Lunatum	F	0	0	32	50	80	91	99	100			•							36
	M	0	8	18	36	64	64	87	98	99	100								42
Naviculare	F	0	0	0	12	30	61	95	99	100									56
	M	0	0	0	4	17	34	51	75	92	99	99	100						72
M. Majus	F	0	0	4	18	53	74	94	99	100									50
	M	0	0	0	4	14	33	51	72	88	96	97	100						72
M. Minus	F	0	0	0	15	40	65	95	100										53
	M	0	0	0	4	22	9	48	81	95	100	99	100						73
Ulnar*	F	0	0	0	0	0	16	60	90	100									68
Epiphysis	M	0	0	0	0	0	7	27	59	82	98	100							80
Pisiforme	F	0	0	0	0	0	0	0	1	19	50	79	96	100					108
	M	0	0	0	0	0	0	0	0	2	6	22	28	66	95	99	99	100	140

*The radial and ulnar epiphyses have been included here because Carter used both in determining the ossification ratio.

†Table I corresponds to Flory's Table V.

The wrist bones are also often relied upon to establish age in a later age group in the period up to near puberty. Table I (taken from Flory's *Osseous Development in the Hand as an Index to Skeletal Development*)[11] indicates the formidable nature of the task for an individual case. The difference due to sex can be as much as 2 years.

Another vital age period in our law is 16 years. The label of this age may, e.g. convert a child into a prohibited immigrant. Reliance is generally placed upon an X-ray examination of the bones forming the elbow joint. Flecker[10] has indicated that there may be a 2-year difference between boys and girls if reliance is placed upon the fusion of the epiphyses with the shafts of the bones—the radiological signs usually relied upon.

It should be quite clear that there is no justification for dogmatism in giving evidence about human age by inference from skeletal studies. The data we have are of statistical value for groups of persons; they have very limited, if any, application to the individual case.

3. The Time of Death

The need to determine the early post-mortem interval arises as a routine in autopsy practice. Indeed, many standard forms of report require this information.

It is generally agreed that the most reliable criteria for this estimation are the stage of rigor mortis present and the temperature of the dead body. It is believed that with the aid of these data, especially the latter, the time of death (in the early stages) can be determined very precisely.

It remains, therefore, to examine these types of observation.

3A. The Development of Rigor Mortis

At the moment of death the body usually becomes completely limp and within a few hours stiffening of the muscles begins and proceeds in an anatomical sequence set out in the standard forensic textbooks with considerable clarity and dogmatism. Indeed, the stage of death stiffening (rigor mortis) reached is employed to estimate the time of death retrospectively.

The classical order in which the muscles stiffen is usually given as follows: The stiffening begins in the muscles of the eyelids and face, then spreads to the lower jaw and the neck, thereafter sweeping symmetrically from the upper part of the body to the four limbs from above downwards. The same order applies to the passing off of death stiffening.

We do not know what the significant chemical and physical changes are which occur in the muscles during death stiffening; but because some of the stiffening muscles surround joints, they immobilize these joints as they become rigid. The fixity of a joint can, therefore, be used as a rough and ready guide to the extent and degree of rigor mortis present. This is a convenient and practical way to make the measurement, but it should be

appreciated that other more precise means of measuring the stiffening of the muscles after death may yet be discovered.

It is difficult to understand why a physico-chemical process which takes place in recently dead tissues should follow the sequence usually described. It appears more likely that, because we are dealing with a physico-chemical process in what is virtually a lump of clay, this will take place *simultaneously* in all the recently dead muscles. If that be the case, it would be reasonable to expect that small masses of muscle would be involved completely much more rapidly than large masses of muscle.

Some support for this hypothesis is to be found in a closer examination of the facts, which forensic pathologists may well in future be advised to claw. I have often observed, in bodies which have not been disturbed and in which rigor mortis had developed to a sufficient degree to involve the wrists and the ankles, that, although there may be no more than about 5 to 10 degrees of movement at the elbow or the knee, there is a much greater degree of movement in flexion or extension at the shoulder or the hip. In other words, although the elbow is almost completely immobilized, the shoulder joint is far from being so.

In addition, it is usually possible to demonstrate a considerable range of adduction, abduction and circumduction in these large joints, as compared with the amount of movement in the elbow and the knee at this stage of the development of the rigor. I have not had an opportunity to determine the precise position in relation to the cervical vertebral joints, but I hope that a re-investigation of the whole problem may be stimulated on a scientific and quantitative basis.

The observations made[12] fit in with the hypothesis suggested, viz. that small masses of muscle would become involved completely more rapidly than large muscular masses; hence a relatively small joint (e.g. the lower jaw joint or the elbow joint) surrounded by a moderate amount of muscle would become immobilized sooner than a large joint, e.g. the shoulder or the hip joint, surrounded by a relatively greater mass of muscle. This might explain the observation that fixity of the elbow or the knee joint occurs to a more advanced degree than of the shoulder or the hip joint at the time of examination. This hypothesis would also explain in part why rigor is first seen in such a small joint as the lower jaw joint.

The account of the invariable progression of death stiffening from the top to the bottom of the body as generally given in the textbooks must, therefore, be modified. The explanation may have to be sought along the lines indicated, viz. that rigor mortis does not progress from the upper end to the lower end of the body in a well-defined fashion, but occurs simultaneously in the recently dead muscles; and that the fixation of a joint depends, among other things, on the involvement by rigor mortis of the quantity of muscle which controls that joint. Variations in the sizes of different joints and in the muscle masses which control them determine

the development of fixation and produce, on superficial study, an apparent top-to-bottom progression of the rigor.

It is well recognized that many environmental and other variables affect the rate of onset, progression and disappearance of rigor mortis. It seems reasonable that a joint which can become immobilized fairly easily by a physico-chemical process because of the relatively small mass of muscle surrounding it, e.g. the temporo-mandibular or lower jaw joint, may also be affected materially by such factors as a raised atmospheric temperature, a humid atmosphere, movement of air if the body is exposed in the open, etc. These and other variable factors will also affect the rigidity of the other joints of the body, but the effects may take longer to develop in joints less exposed to these factors.

If the hypothesis advanced to explain these observations is more in keeping with the facts observed, it is obvious that a still more limited reliance than is already the case at present must be placed upon data dependent upon the distribution of rigor mortis in a dead body for the purpose of estimating the early post-mortem interval.

It remains a matter for remark that, from edition to edition, textbook after textbook has studiously and uncritically been copying inaccurate descriptions of one of the most basic and fundamental observations to be made on the dead. It is clear that forensic pathology cannot subscribe (in the words of Sir Thomas Browne) to a 'peremptory adhesion unto authority'. Here indeed is need to re-examine many tenets held dear by the uninquiring mind for many generations.

To the taking of thought upon this subject should be added the fructifying act of the laying on of hands.

3B. Allegations about the Rate of Cooling of the Dead Body

At the moment of death the complicated physiological mechanism whereby the heat of the body is kept constant, is destroyed. The healthy human body may then be regarded as a lump of clay more or less uniformly heated to 98.4° F and which will progressively lose heat according to physical laws until its temperature becomes the same as that of its environment. Many variables influence the rate at which this cadaveric cooling occurs, but our concern is limited to primary factors inherent in the corpse itself. We will not deal with the environmental influences affecting the rate of cooling. These, though very inexact, are not the subject of controversy.

The post-mortem temperature of the body is generally regarded as one of the most valuable observations from which the time of death can be calculated in retrospect. Keith Simpson[6] states that the cooling of the body is the 'only reliable indication of the lapse of time during the first 18 hours after death, and its *early* measurement is often vital to the establishment of the exact time of death'. This view is widely accepted (especially

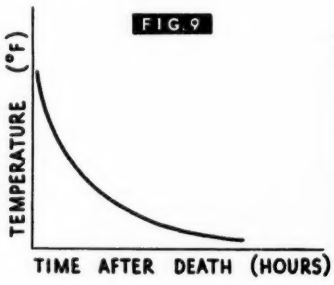
by novelists) and many empirical rules of thumb have been enunciated to simplify the calculations involved. Keith Simpson, e.g. states (*op. cit.*, p. 6):

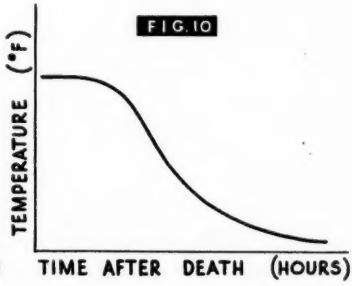
'Under average conditions the clothed body will cool in air at the rate of about 2½° F. an hour for the first 6 hours and average a loss of 1½–2° for the first 12 hours . . . the temperature of the internal organs should be the same as the environment in 20–24 hours.'

Rhodes, Gordon and Turner[7] (*op. cit.*, p. 75) state:

'At an atmospheric temperature of about 60° F. the clothed and well-developed adult takes about 24 to 30 hours to cool to the temperature of the environment. Cooling, under these conditions, occurs at the rate of about 1½° F. per hour for the first 12 hours and at a rate of about 1° F. per hour for the succeeding 12–18 hours.'

Both these authorities depict the rate of cooling by the same type of graph (Fig. 9).





It is probably true that the temperature of the body is the most reliable guide we have to the time of death; but whether, from the data available, the time of death can be determined so exactly as some forensic pathologists would have us believe, is extremely doubtful. The careful observer will certainly find that actual temperature measurements on the recently dead are in conflict with the results obtained by the use of the empirical rules laid down.

To begin with, in many books the method of measuring the temperature is not given. The acceptable technique is to insert the special thermometer available for this purpose into the interior of the belly cavity under the liver. If this is done, disconcerting results are often obtained in conflict with what one would expect if an attempt is made to interpret the results in terms of the type of graph shown in Fig. 9.

Fig. 9 is probably an accurate representation of what happens to the temperature of a small inorganic body which undergoes cooling, but the curve does not fit what happens to the human corpse, when the temperature is measured in the recommended way. The human cadaver may be a lump of clay, but it is a lump of considerable mass and of a particular shape.

The problem may be simplified by considering what happens when a solid sphere of uniform temperature cools to the temperature of its environment. From the centre of the sphere heat is lost by conduction to the

surface, from where further heat loss occurs to the atmosphere by radiation and convection.

The rate of cooling will depend, *inter alia*, on the heat conductivity of the material. Elaborate mathematical calculations can be made for the rate of cooling at points along the radius of the sphere. The surface will, at the beginning, cool more rapidly than the centre of the sphere, because of the greater temperature difference. Moreover, because the dead human body is a poor conductor of heat, it is to be expected that a similar state of affairs will take place in the human cadaver in the early hours after death. As the temperature is measured in the interior of the belly cavity, there will be no measurable change in the early hours after death. It was in an attempt to apply contemporary views on the cooling of dead human bodies to estimate the time of death that I met with considerable difficulty. I repeatedly came across cases in which apparently healthy persons who had died at known times were found to have normal temperatures for at least 4 hours after death, when, according to the popular rules of thumb, the temperature should have dropped by some 10° F.

The analogy of what happens to the cooling sphere helps us to understand the position. As the rate of cooling of the interior of a solid body will depend on conduction, the amount of heat at the centre of a large sphere or ovoid cylinder (which the human frame virtually is) can be expected to remain unchanged for some time. We know from actual observation that this period may be at least 4 hours. It is possibly considerably longer. A more adequate graphic representation of the way in which the dead body cools is submitted in Fig. 10. The initial plateau corresponds with what is observed in nature and illustrates immediately the very great difficulties which may be encountered in relying upon the temperature of a dead body in the early hours after death in order to establish the time of death. It would be most unwise to rely entirely upon this single factor. It cannot be invested with that certainty to which some forensic pathologists are apparently partial and prone.

A Note on the Estimation of the Time of Death

Interesting developments are taking place which may provide more reliable ways of estimating the time of death in adults, if this has occurred within 15 hours. The basic work has been done by Schourup in Denmark and is available to English readers in that excellent monograph, edited by Dr. Keith Simpson, entitled *Modern Trends in Forensic Medicine*, 1953, p. 80, where Dr. Mant surveys this field. Schourup's work will need extension and confirmation, but it strongly suggests that a chemical examination of the lactic acid, non-protein nitrogen and the amino acid content of the cerebrospinal fluid may be more helpful than any of the other observations heretofore relied upon. Schourup used these chemical analyses to calculate the time of death in 50 cases. His results were:

Exact time calculated	34%;
Exactness to ± 1 hour	84%;
Exactness to ± 1½ hours ..	100%.

Mant summarizes the position by saying that in adults (but not in children) this method offers a 3-hour margin up to 15 hours after death.

4. Specific Tests for Human Blood

The field of forensic pathology is redolent of myth. Unverified allegations continue to be copied uncritically from textbook to textbook and are almost literally handed down from father to son. The province of serology is no exception to this rule, and major unwarranted assumptions about the specificity of the tests for human blood continue to be made. Modern serological techniques permit the revaluation of the adequacy of dogmas based on an inadequate appreciation of the limitations of the routine laboratory.

For these reasons the quantitative work reported by Dr. J. E. Duncan Taylor[13] has a special importance, particularly in South Africa, where the problems created by a possible confusion with non-human primate blood may be very real. Dr. Duncan Taylor's researches were carried out in Durban as a member of the staff of the Pathological Laboratory of the Union Health Department—a laboratory which has in recent times made distinguished contributions to medico-legal research.

The present issue is essentially a practical one because a decision affecting the life of an accused person may depend on the report of the forensic serologist, yet it is not generally appreciated that Man shares so closely with other primates a serological reaction to anti-human serum prepared in rabbits, that the test-tube reactions cannot be differentiated by the usual routine methods described in current medico-legal textbooks.

Dr. Duncan Taylor's work is a thorough survey of contemporary methods and demonstrates their inadequacy because of their non-specificity. The comprehensiveness of her research is reflected in the considerable range of animals tested, extending throughout all the phyla from fishes to birds.

One conclusion emerges clearly from her survey, viz. that there is no simple, practical method available for laboratory testing which can distinguish human blood stains from the blood stains of apes, monkeys (including baboons) or lemurs, e.g. the nagapie.

The medico-legal implications of this state of affairs are obviously very important. Her excellent investigation has made it clear that the greatest caution must be exercised in framing reports intended for our Courts so as not to create an impression that the tests have definitely proved a stain to be human. An expert witness who (basing himself on the current routine laboratory methods of investigation) dogmatically asserted that a blood stain was in fact of human origin, could undoubtedly be cross-examined very severely and devastatingly and find himself unable to maintain the dogmatic assertion that the blood stain was exclusively of human origin. It is unlikely that he can go beyond classifying the stain as belonging to the primate group, i.e. Man, the higher anthropoid apes such as the gorilla, the gibbon, the chimpanzee, the orang-outang; and monkeys, baboons and lemurs.

From Dr. Duncan Taylor's work it is clearly inaccurate and misleading

even to report that a blood stain is of 'human or anthropoid origin'. The term 'anthropoid' means resembling *anthropos* or Man; its use is, therefore restricted to those tailless apes which resemble Man. Dr. Taylor's work exposes this vulgar error. Such inexactitude in terminology should be avoided rigorously as it reveals a lack of acquaintance either with the serological problems involved, or with zoological nomenclature.

Although it is clear from Dr. Duncan Taylor's survey that no simple practicable method is available for routine testing, the important fact emerges that where the gravity of the case warranted the trouble, the specific identity of a particular stain probably could be established. The technique required, however, is elaborate, expensive and time-consuming, and would mean that the appropriate primates would have to be inoculated in order to obtain the specific anti-sera for performing the tests.

Apart from the medico-legal importance of this kind of work, it obviously has considerable significance for anthropological and evolutionary studies. It emphasizes, moreover, the great care, knowledge, skill and caution needed to carry out this kind of investigation and to give expert evidence about it.

5. Does Arsenical Poisoning Preserve the Dead Body?

One of the most sedulously cultivated medico-legal superstitions, unsupported by anything except mere allegation, is that fatal arsenical poisoning prevents putrefaction. Glaister[11] says that the bodies of those who have died from poisoning by arsenic resist putrefaction longer 'owing to the preservative action of such substances on the tissues, or to their destructive, or inhibitive action on the organisms which induce decomposition'. Glaister also states (*op. cit.*, p. 547):

'The preservative influence of arsenic upon the tissues of those poisoned by this substance has been repeatedly observed and noted following exhumation, despite assertions to the contrary'.

Smith [11] distinguishes between acute and chronic arsenical poisoning and states:

'In death from acute arsenic poisoning, the stomach may be fairly well preserved. . . . In chronic poisoning . . . there may be a distinct retardation of putrefaction, especially if the poison has been given in small doses over a long period.'

The latter is a difficult argument to follow, as arsenic is constantly being excreted during the chronic form of poisoning and the amounts found in the viscera are usually less than in the case of acute poisoning.

The authorities are formidable and their views were certainly held by that redoubtable witness, Sir Bernard Spilsbury, who had no reservations in his mind about fatal arsenical poisoning as a cause of the preservation of the corpse. In the well-known Seddon case, Spilsbury was prepared to admit, on the basis of his autopsy findings, that death could have been due to 'epidemic diarrhoea', but he excluded this probability because of the condition of the preservation of the body.[14]

A valuable South African contribution to the solution of this difficulty has been made by Copeman and Kamerman.[15] They addressed themselves to this problem. Their data make it clear that putrefaction proceeds despite fatal arsenical poisoning and that in the few cases where moderate or good preservation of the corpse was found, the physical conditions of the soil, its drainage, etc., provided an adequate explanation of what was observed. Their conclusions were based on the study of 35 cases of fatal arsenical poisoning during a period of 5 years. Only 3 of the cases showed any state of preservation and of these, one case 'was extraordinarily well preserved, even after a year's burial. As, however, the evidence showed that before death the deceased vomited extensively, and as it is known that the cemetery in which the deceased was buried is very well drained, the cause of the preservation may well be sought elsewhere than in the presence of arsenic in the body.'

It is, of course, also a matter of practical experience that bodies not dead from arsenical or other forms of poisoning may be remarkably well preserved, even for many months.

It is in the nature of things difficult to assess quantitatively the factors which influence the changes occurring after burial, e.g. the presence of the coffin, the clothing or other coverings, the depth of the burial, the drainage of the soil, access of air to the body after burial, the effect of physical contact between dead bodies in mass graves—all factors which have been the subject of an interesting survey by Mant.[81]

The available evidence thus makes it necessary to sound a note of warning and caution about placing any reliance on the modest amount of arsenic which can be fatal, as a cause of the preservation of the dead body. The views expressed by Copeman and Kamerman can be endorsed strongly. We should also bear in mind their statement that comparatively large amounts of arsenic are used in embalming, thus making it difficult to see how the amount of arsenic usually found in the organs in fatal arsenical poisoning can have any significant effect in preventing decomposition.

There seems no basis for arguing in converse that, because the body has been well preserved, this is evidence in favour of fatal arsenical poisoning. The clinical history of the case, the almost negative autopsy findings and the toxicological demonstration of arsenic in the appropriate viscera or tissues constitute the only kind of evidence upon which a medical witness should be prepared to rely.

6. The Misuse of the Term 'Cyanosis'

The next fallacy to which I invite your attention arises from the misuse of the clinical term 'cyanosis' in descriptions of the skin colour of the dead. 'Cyanosis' means 'blueness' and the word was coined to describe an appearance of the surface of the body due to certain changes in the oxygen-carrying capacity of the blood.

Oxygen is carried in the blood in a fairly loose combination with an iron compound (the red colouring matter of the blood) called haemoglobin. When the iron compound in the blood takes up the oxygen, it does so by a chemical process which is the same as that involved in rusting. When this 'rusted' blood reaches the tissues of the body and gives up its oxygen, it does so by a process of 'unrusting'; then it loses its bright red (rusted) colour and becomes a purplish blue. When there is a sufficient amount of this 'unrusted' blood in the arterial side of the circulation, the skin will take on the blue colour of the 'unrusted' blood. Normally, the contribution which the blood makes to the colour of the skin is by virtue of the presence of 'rusted' blood in the ultimate sub-divisions of the arterial tree, viz. the capillaries. Consequently, when this 'blue' 'unrusted' blood circulates in the surface capillaries the patient's appearance also becomes bluish and can properly be described as cyanotic. This is abnormal.

Anything, therefore, that interferes with the oxygenation (rusting) of the blood, e.g. in the lungs (or that short-circuits blood from the venous to the arterial circulation) will produce an appearance of cyanosis, particularly of the ears and the lips and the tips of the extremities, sites in which the cyanosis is usually detected very easily.

We need not consider the numerous causes of cyanosis, but the fundamental principle involved in its production should be understood clearly, because to diagnose cyanosis correctly is to appreciate certain important physiological possibilities underlying this appearance.

In the dead no dynamic chain of events such as I have described can take place. The arterial blood all over the body (including the capillaries) will begin to 'unrust', i.e. to yield up the oxygen loosely combined with the haemoglobin. But the whole animal body does not die at a particular moment in time. The precision of the moment of legal death is a fiction devised to satisfy the arbitrary requirements of the legal mind. Biologically we die in bits and pieces, and tissue respiration continues for a short time after legal death can be certified. 'Unrusting' (oxygen dissociation) therefore continues in the recently dead or dying tissues and this process will go on until equilibrium has been reached between the tension of the oxygen in the capillaries and that in the neighbouring tissues.

In addition, there may be reflux of venous 'unrusted' or 'blue' blood from the venular end of the capillaries, thus adding to the blueness of the blood *post mortem*.

For these reasons the colour of the blood in the dead becomes purplish blue and not as a result of some interference with the physiological processes whereby oxygen is bound to the haemoglobin of the blood in the lungs, e.g. throttling, strangling, drowning, etc.

A second important point to consider is that, as a result of the force of gravity, after death the liquid blood gravitates to the most dependent parts, a phenomenon known as the development of post-mortem lividity.

(*The Concise Oxford Dictionary* defines 'lividity' as 'of bluish leaden colour'. This is a very accurate, non-committal and objective description of the appearance commonly seen in the recently dead.) Two factors are, therefore, important.

1. The post-mortem diffusion of oxygen gas until equilibrium has been reached with the neighbouring tissues (together with possible venular reflux) will produce a blood bluish in colour irrespective of the cause of death. (We except certain special instances such as carbon monoxide poisoning, prussic acid poisoning, etc., which do not concern the point at issue.)
2. By the physical law of gravitation this bluish coloured blood will gravitate into the dependent parts of the body.

The supine position is the commonest in which dead bodies are found and the position of the hands is such that the finger tips are also usually in the dependent position. The head may or may not be turned to one side and the result established in the earlier hours after death is a distribution of bluish coloured blood into the very areas in which cyanosis is looked for in the living, viz. the face, the ears, the tips of the fingers.

The detection of bluish coloured blood in these situations is not, therefore, to be regarded as evidence of the manner in which the deceased person met his death. The pathologist is inspecting no more than the normal appearance of post-mortem lividity and he can easily be misled into making major assumptions about the cause of death if, without any justification, he infers that the bluish colour of post-mortem lividity has the same connotation as cyanosis produced during life. It is not uncommon to find in post-mortem reports rendered by district surgeons a statement that there was *cyanosis* of the face and the finger nails, because of this confusion between cyanosis and lividity.

In a recent South African case of alleged asphyxial death (submersion and throttling) the district surgeon described in his autopsy report that the face and the finger nails of the deceased were cyanosed (blue). At the preparatory examination he argued from this evidence that the word 'cyanosed' used in the post-mortem report indicated that the nails and the face were blue and that this in turn indicated asphyxia. This mental process may well reveal an inarticulate major assumption in the mind of the district surgeon about the cause of death.

It is quite clear that evidence about cyanosis, if the word is used in its proper clinical sense, must come from other quarters (i.e. the practitioner who examined the patient when he was alive) and not from the pathologist or the district surgeon performing an autopsy.

As it is not possible to distinguish the discoloration seen in dead bodies from that produced by a cyanotic process in the living, the use of cyanosis to describe appearances in the dead is strongly to be deprecated.

Taylor's *Principles and Practice of Medical Jurisprudence*,[16] gives the following list of synonyms for post-mortem lividity (p. 181):

'*post-mortem* hypostases, subcutaneous hypostases, cadaveric lividities, suggilations, vibices, *post-mortem* stains.'

It is noteworthy that 'cyanosis' is excluded and that it is not used either by Smith or by Glaister as an alternative to 'hypostasis' or 'lividity' in the chapters of their textbooks dealing with post-mortem lividity; and Polson, Brittain and Marshall[23] also avoid the use of the term 'cyanosis' when dealing with the post-mortem appearances of the dead.

As we now know, the *normal* colour of areas of post-mortem lividity is a 'cyanotic' hue and this appearance will, therefore, be seen in areas such as the face, the ears and dependent extremities such as the finger tips. It is commonplace to see so-called cyanosis under the finger nails at autopsy even in cases of death not due to asphyxia resulting from obstruction of the airway. The use of the term 'cyanosis' in such circumstances reveals an entirely unwarranted presumption about the cause of death and a prejudice in favour of a diagnosis of asphyxia. It is unjustifiable to invest a non-committal, chromatic appearance with such a diagnostic significance which the observation must lack because of the non-specific way in which the bluish colour of the blood is brought about after death.

Evidence about cyanosis must be adduced from other sources than those of a post-mortem inspection several hours after death. I need hardly emphasize that the fallacious inferences which may otherwise be drawn from what is a normal development of post-mortem lividity may be prejudicial to the course of justice.

7. The Liquidity of the Blood in Asphyxia

Many reputable reference works on forensic pathology continue to perpetuate the myth that the blood remains liquid longer in asphyxial deaths and that this is a valuable autopsy sign confirming the view, in a particular case, that death was due to some mechanical interference with the ability of the tissues to use oxygen.

It is also generally claimed that this increased liquidity of the blood, due to a prolonged clotting time, results from the increased amount of carbon dioxide which accumulates in the circulation during asphyxia or, as it should more properly be called, anoxic anoxia.

Eminent exponents in the forensic field have long been a party to the dissemination of these views. Indeed, as recently as 1936, in the trial of Dr. Buck Ruxton for the murder of his wife and a servant, no less an authority than Prof. John Glaister expressed the opinion that in 'asphyxial death there is an increased interval of time during which fluidity of blood may remain—up to 12 hours or thereabouts, but I would not be certain'.[17]

Taylor,[16a] who is recognized in many countries as the supreme authority in matters of medical jurisprudence, states (in connexion with the post-mortem appearances of simple asphyxia) that 'the blood is found fluid for

an unusually long time after death; it coagulates (i.e. clots) very slowly, owing amongst other factors, to excess of carbonic acid (CO_2) contained in it, and for the same reason is very dark in colour'.

That fluid blood has been observed *post mortem* in cases of asphyxial death will not be disputed by any medico-legal pathologist of any experience; but there is every reason to doubt whether this fluid blood is a sign pathognomonic of asphyxia as the cause of death. Those who are beguiled by what Sir Thomas Browne has aptly called a 'peremptory adhesion unto authority', will readily recall the presence of this same kind of liquid blood in cases in which death was clearly not due to asphyxia. While the observation of fact is not doubted, the interpretation which has been placed upon it is certainly very suspect. No difference in clotting time is recognized between *normal* venous and arterial blood. This alone should have made the medico-legal experts cautious. Moreover, in recent years the matter has been put to the text of experiment and the orthodox views have certainly been found wanting. Ponka and Lam, who produced asphyxia in dogs under experimental conditions, concluded that severe asphyxia did not have any effect on the clotting properties of canine blood. 'There was certainly no tendency for the second specimen, obtained during asphyxia, to show a prolongation of clotting time which would have indicated a breakdown of the coagulation mechanism.'[18]

A very adequate review of the whole problem has been published by Mole[19] who states: 'Emphasis on the fluidity of the blood as characteristic of any special cause or mechanism of death is probably misplaced . . . ' These conclusions are in accord with the earlier observations reported by the Russian investigator Yudin[20] in his work on the use of cadaver blood for transfusion and confirms what Hunter wrote as long ago as 1794: 'In many modes of destroying life the blood is deprived of its power of coagulation, as happens in sudden death produced by many kinds of fits, by anger, electricity or lightning; or by a blow on the stomach, etc. In these cases we find the blood, after death, not only in as fluid a state as in the living vessels, but it does not even coagulate when taken out of them.'[19]

Mole concluded that the liquidity of the blood was a feature of 'sudden' death associated with 'shock or collapse' during life. The mechanism appears to be the production of a fibrinolysin. In certain deaths associated with infection and cachexia, this fibrinolysin may fail to develop, thus explaining the presence of abundant clot in the heart as well as in the limb vessels in such cases.

Mole also makes the important observation that 'fluid and coagulable blood was always found, whatever the cause of death, when the autopsy was carried out within an hour or so of death'. It is clear that in cases of 'sudden' death the blood remains spontaneously coagulable 'only during a brief period after death and then becomes completely free from fibrinogen' (Mole, *op. cit.*, p. 419); hence it will never again clot, whether asphyxia has

contributed to the fatal outcome or not. Indeed, this incoagulability of the blood is a commonplace at autopsy and the finding of clots is the unusual thing.

The modern approach to the problem of liquid post-mortem blood has also been appreciated by Camps. Camps gave evidence in the case in which Donald Hume pleaded guilty to being an accessory after the fact in connexion with the murder of Stanley Setty, whose torso was disposed of by being dropped into the sea from an aeroplane.

Camps, recognizing that the blood of the cadaver could remain permanently liquid, states: 'This must modify previous teaching that clotting is complete after death in 12 hours.'[21]

It seems that many medico-legal pathologists have mistakenly come to regard the common occurrence of the liquid and incoagulable blood found at autopsy in a great variety of different kinds of death as a diagnostic sign of the anoxic type of death occurring in asphyxia. This completely disregards the properties of cadaver blood, which are in great measure determined by the post-mortem interval. This erroneous view has been perpetuated from textbook to textbook, and the time for its correction is certainly overdue. We can no longer base our opinions on the arbitrary and undisciplined dictates of antiquity.

8. Some Conclusions About Vulgar Errors

This octette of cameos reveals a disquieting state of forensic affairs. Many hoary observations claimed as facts are not verifiable by experience. They seem, on the contrary, to depend on arbitrary assumption or inadequate authority.

The spell-binding influence of the printed word may provide an explanation (but certainly no excuse) for this dangerous and uncritical attitude reminiscent of the paralysing grip which the sterile mediaeval tradition imposed on medical and scientific knowledge in its time—a tradition which sacrificed observation to occult speculation and which set more store by authority than experiment.

There is clearly need for fundamental research in the field of medico-legal pathology, particularly as the data required are not only of academic interest or importance. They touch the very lives of our fellow men.

Although the examples surveyed cover only a small portion of a very suspect field of knowledge, they should serve as a guide to the type of inquiry indicated and the need for a rigorous application of the accepted and tested criteria of experiment and controlled observation evolved and vindicated by the methods of science.

I have touched on so-called cyanosis observed in the dead body and the alleged liquidity of the blood in asphyxia as two of the criteria commonly relied upon for the crucially important medico-legal diagnosis of asphyxia.

The diagnostic problem (of establishing the cause of death) raises, of course, very great difficulties. These have been exposed and clarified by a particularly acute analysis published by Gordon,[22] who has suggested a more adequate and helpful classification of deaths of forensic importance than has been available hitherto.

I have already illustrated the errors which follow the unjustified application of a clinical nomenclature to describe a different order of things in the dead. It seems to me, by the same argument, that much confusion has arisen in the past because of the failure to appreciate that coma, syncope and asphyxia are not pathological entities which can be recognized as such by an inspection of the viscera of a corpse. They are clinical entities whose diagnosis depends on clinical techniques of examination.

Gordon's hypothesis is based on verifiable observation and is coherent with the established body of physiological and pathological knowledge. His classification is important because it draws attention implicitly to this distinction and imposes some intellectual discipline and order on what might well be described as clinico-pathological chaos in this field. It is therefore, important to divorce those non-specific signs common to almost all forms of death, however the ineluctably fatal sequence of events has been set in motion, from the specific signs which may (in certain limited instances) give the medico-legal pathologist some clue to the likely mode of death in a particular case, e.g. asphyxia due to throttling.

It is essential that unbiased and unprejudiced attitudes should be brought to bear upon the study of the dead body. We must recapture the scepticism of Vesalius, who was not content to accept the writings of Aristotle or Galen merely because of their awesome authority. Like him, we must put everything to the ultimate test of experience. Otherwise the modern pathologist may have to be equated with the ancient Roman augur who, with prophetic insight, offered his soothsayer's opinion after inspecting the entrails of a corpse. It is a matter of some concern that many a forensic opinion in medico-legal pathology has virtually not advanced beyond the stage of such Roman quackery.

It may, indeed, be a matter of regret that biological phenomena are not always capable (because of their intrinsic variability) of providing that precise answer, with qualities of papal infallability, which legal questions so often demand. But this is not the fault of the medical witness, who must consequently guard with great care against an omniscience and a positiveness which, in the nature of things, he cannot have. His claims in such directions would merely demonstrate that he stands alone in the possession of unique talents, which pathologists in the rest of the scientific world lack. It is as well to remind ourselves of the pertinent statement by Sir Thomas Browne (from his *Pseudodoxia Epidemica*), now some 300 years old, which reads:

'But the mortallest enemy unto knowledge, and that which hath done the greatest execu-

tion upon truth, hath been a peremptory adhesion unto authority; and more especially, the establishing of our belief upon the dictates of antiquity. . . . Now hereby methinks we manifestly delude ourselves, and widely walk out of the track of truth'.

References

1. Smith, S. (1945): *Forensic Medicine*, 8th ed., p. 344. London: J. & A. Churchill, Ltd.
1a. Smith, S. (1949): *Forensic Medicine*, 9th ed., p. 32. London: J. & A. Churchill, Ltd.
2. Shapiro, H. A. (1947): *The Limited Value of Microscopy of Lung Tissue in the Diagnosis of Live and Still Birth*, Clin. Proc., **6,** 149.
3. Gibberd (1943): *Short Textbook of Midwifery*, 3rd ed., p. 497. London: J. & A. Churchill, Ltd.
4. Glaister, J. (1950): *Medical Jurisprudence and Toxicology*, 9th ed. Edinburgh: E. & S. Livingstone, Ltd.
4a. Glaister, J. (1950): *Ibid.*, p. 139.
5. Ham, A. W. and Baldwin, K. W. (1941): *A Histological Study of the Development of the Lung with Particular Reference to the Nature of the Alveoli*. Anat. Rec., **81,** 363.
6. Simpson, C. Keith (1952): *Forensic Medicine*, 2nd ed., p. 24. London: Edward Arnold & Co.
7. Rhodes, W. F., Gordon, I. and Turner, R. (1945): *Medical Jurisprudence*, 2nd ed., p. 186. Cape Town: Stewart Printing Co. (Pty.), Ltd.
8. Simpson, C. Keith, Ed. by (1953): *Modern Trends in Forensic Medicine*, p. 13. London: Butterworth & Co., Ltd.
8a. Mant, R. (1953): *Ibid.*, pp. 84 *et seq.*
9. Pryor, J. W. (1928): *Differences in the Ossification of the Male and Female Skeletons*, J. Anat., **62,** 499.
10. Flecker, H. (1932): *Roentgenological Observations of the Times of Appearance of the Epiphyses and their Fusion with the Diaphyses*, J. Anat., **67,** 118.
11. Flory, C. D. (1936): *Osseous Development in the Hand as an Index of Skeletal Development*, Monographs of the Society for Research in Child Development, Vol. I, No. 3. Society for Research in Child Development, National Research Council, Washington, D.C.
12. Shapiro, H. A. (1950): *Rigor Mortis*, Brit. Med. J., **2,** 304.
13. Taylor, J. E. D. (1952): *The Medico-Legal Precipitin Test for Human Blood*, S. Afr. Med. J., **26,** 81.
14. Young, Filson, Ed. by (1952): *The Trial of the Seddons*, 2nd. ed., p. 103. Notable British Trials Series, London: Wm. Hodge & Co., Ltd.
15. Copeman, P. R. van der R. and Kamerman, P. A. E. (1940): *Poisoning by Arsenic in South Africa*, S. Afr. Med. J., **14,** 379.
16. Taylor (1948): *Principles and Practice of Medical Jurisprudence*, Ed. by Sir Sydney Smith and W. G. H. Cook, Vol. I, 10th ed., p. 181. London: J. & A. Churchill, Ltd.
16a. Taylor (1948): *Ibid.*, p. 520.
17. Blundell, R. H. and Wilson, G. H., Ed. by (1950): *Trial of Buck Ruxton*, 2nd ed., p. 157. London: Wm. Hodge & Co., Ltd.
18. Ponka, J. L. and Lam, C. R. (1948): *Effect of Asphyxia on Blood Coagulation in Dogs*, Proc. Sc. Exp. Biol. Med., N.Y., **68,** 334.
19. Mole, R. H. (1948): *Fibrinolysin and the Fluidity of the Blood Post Mortem*, J. Path. Bact., **60,** 430.
20. Yudin, S. S. (1937): *Transfusion of Stored Cadaver Blood*, Lancet, **2,** 361.
21. Camps, F. E. (1951): *The Case of Stanley Setty*, Med.-Leg. J., **19,** 2.
22. Gordon, I. (1944): *A Classification of Deaths of Medico-Legal Importance*, Brit. Med. J., **2,** 337.
23. Polson, C. J., Brittain, R. P. and Marshall, T. K. (1953): *The Disposal of the Dead*, pp. 222, 257. London: English Universities Press, Ltd.

MEDICO-LEGAL CASE BOOK

2: Yeasts and the Production of Alcohol Post Mortem

Harald Gormsen, M.D.
University Medico-Legal Institute, Copenhagen

At the University Institute of Forensic Medicine in Copenhagen we have for a good many years systematically performed analyses of post-mortem blood in connection with all autopsies on traffic and accident victims, all poisoning cases and in a great number of cases of suicide and sudden natural death. I should like to mention a few of our experiences, especially concerning the sources of error.

In Denmark we use as a routine for alcohol estimations the Swedish Widmark method, a method very much like the Niclous method, i.e. involving the reduction of potassium dichromate.

If a moderate or pronounced putrefaction of the blood has occurred, values that are too high will be obtained by this method because of volatile reducing substances in the putrid blood. The urine values will be more valid in such cases, because no reducing putrefaction products will appear in the urine, or only to a much smaller extent.

In all cases in which we deal with putrefaction we use (besides the Widmark method) the almost specific Zeisel method. This involves treatment with hydriodic acid and subsequent distillation of the volatile ethyl iodide into a solution of silver nitrate. The precipitate found is dried and weighed.

Very recently also the enzymatic ADH (alcohol dehydrogenase) method has been used in collaboration with the Stockholm Forensic Toxicology Laboratory.

By these very exact and almost specific techniques we are able to check the values found with the routine reduction method of Widmark.

The nature of substances in putrid blood giving rise to the apparently false alcohol reaction by the reduction methods is obscure, but I shall mention a definite source of error in which there is actually a post-mortem *production* of alcohol.

We recently had a peculiar case of natural death in a neurasthenic 43-year-old woman. One of our assistant doctors who did the autopsy 20 hours *post mortem* in a provincial hospital came back to the Institute and reported

that he had found no definite cause of death, 'but she had lots of port wine in the pleural cavities and in the stomach contents'. The blood examination gave no significant alcohol reaction (only 0·02%) and I was inclined to think that the assistant doctor himself possibly had had port wine in his stomach contents.

However, alcohol examinations (by all the 3 methods mentioned) showed 0·3% alcohol in the pleural exudate and 0·23% alcohol in the stomach contents.

Bacteriological examinations of the pleural exudate and the stomach contents showed lots of fungi (*Candida albicans*=thrush fungi) and yeast cells (Saccharomyces); both these micro-organisms were found to be able to produce alcohol in glucose bouillon *in vitro*.

By interviewing the relatives and the doctors in the case mentioned it was definitely excluded that the patient had had any alcohol for several days before death occurred. She was *not* a diabetic. The urine taken at the autopsy gave no reaction for sugar.

We had to conclude that the alcohol content in the pleural fluid and in the stomach was due to post-mortem spreading of the micro-organisms mentioned, and was produced *post mortem* by these micro-organisms.

After the shock we got from this case, we feel obliged to do a bacteriological examination, in order to establish whether fungi or yeast cells are present in the material examined, in all cases in which the result of the post-mortem alcohol analysis is of decisive importance.

Pharmacological Aspects of Barbiturate Intoxication*

E. J. Wayne, M.D., Ph.D., F.R.C.P.

Department of Pharmacology and Therapeutics, The University of Sheffield

I propose to deal with certain selected topics which will I hope be of special interest to you. Naturally, we look at the barbiturates from a different point of view. When I think of them there comes to my mind their chemical formulae, some thoughts on proprietary versus official preparations, the frequency with which phenobarbitone is used as a placebo and the memory of many patients admitted to my wards unconscious but usually responding well to treatment.

It is because the speed of onset of action and the duration of action can be varied both by the choice of a particular barbiturate and by altering the dose that these drugs are so widely used.

The barbiturates depress the central nervous system with remarkably little effect on the circulation or metabolism, but with a pronounced tendency to cause respiratory depression in full doses. They are rapidly and completely absorbed by the oral route, the sodium salts being rather more quickly absorbed. Barbitone is excreted unchanged in the urine, as is a substantial fraction of the dose of Phenobarbitone, Dial and Rutonal. The other barbiturates are oxidized mainly in the liver and do not appear in the urine. It is consequently dangerous to use the short-acting barbiturates in liver disease and the long-acting barbiturates in renal disease.

Alcohol and Barbiturates

One aspect of the action of barbiturates which is of special importance and which has been fully studied in animals is the effect of combining alcohol and the barbiturates. It has been shown by several authors that alcohol potentiates the action of barbiturates and that both the lethal dose and the anaesthetic dose of a barbiturate are less if alcohol is present in the blood and tissues. In man, Jetter and McLean[1] have described 3 cases in which the cause of death appeared to be the combined actions of a barbiturate and alcohol, and Fisher *et al.*[2] have reported blood concentrations not usually fatal in cases dying from barbiturate poisoning with high blood alcohol levels. Thus one case with a blood alcohol of 0·17% had a barbiturate level

* Read at a meeting of the British Association of Forensic Pathologists in November 1952 (London).

of only 0·6 mg. per 100 c.c., a level which may result from as little as 0·6 gm. (9 grains) of Seconal. Morphine also potentiates the action of barbiturates on the respiratory centre.

Dose Variation

The next point to which I would draw your attention is the great variation in the dose of a barbiturate required to give the same response, e.g. anaesthesia. Fig. 1 shows that the mean narcotic dose of Sodium Amytal is about

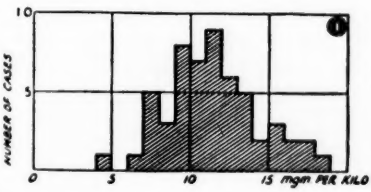


Fig. 1. Dosage of sodium amytal (mg. per kilo) needed to produce adequate narcosis when given by slow intravenous injection to 55 obstetric cases (Paxson[3] 1932).

11 mg. per kg., but the range is from 4 to 18 mg. per kg. With most drugs there is, in a group of 100 persons, at least a fourfold difference between the dose required to give the same effect in the least and the most susceptible individual. For this reason pharmacologists rarely talk of minimum lethal doses, but usually of the LD50, i.e. the dose which will kill 50% of a population. Attempts have been made to calculate this figure in the case of the barbiturates in man by collecting statistics from hospitals. Table I shows

Table I

Drug	*Fatal Doses*		
	Minimum	*Median*	*Maximum*
Barbitone	2.0 G. (30 gr.)	6.0 G. (90 gr.)	24.0 G. (360 gr.)
Phenobarbitone ..	1.6 G. (25 gr.)	5.25 G. (80 gr.)	17.5 G. (280 gr.)
Amylobarbitone ..	—	1.6 G. (25 gr.)	—
Pentobarbitone ..	—	?1.0 G. (15 gr.)	—
"Seconal" ..	—	?1.2 G. (18 gr.)	—
	Non-Fatal Doses		
	Minimum	*Median*	*Maximum*
Barbitone	0.3 G. (5 gr.)	33. G. (50 gr.)	32.5 G. (500 ge.)
Phenobarbitone ..	0.2 G. (3 gr.)	1.7 G. (26 gr.)	9.1 G. (140 gr.)
Amylobarbitone ..	0.2 G. (3 gr.)	—	10.0 G. (150 gr.)
Pentobarbitone ..	0.8 G. (12 gr.)	—	8.0 G. (120 gr.)
"Seconal"	0.75 G. (11 gr.)	—	5.0 G. (90 gr.)

the sort of figures which result. The median fatal dose is inaccurate in the case of the rapidly-acting barbiturates and the non-fatal doses are more accurate. In general, the dose causing severe symptoms is 5 to 10 times and the fatal dose is about 15 times the usual oral dose.

The therapeutic index of some of the barbiturates has been determined in animals, i.e. the ratio LD50 : AD50 (lethal to anaesthetic dose). Seconal, Amytal and Pentobarbitone (ratio 2·5, 2·4 and 2·2) appear to have a bigger margin of safety than Barbitone (1·41) and Phenobarbitone (1·66), at least in animals.

Symptomatology

The common symptoms of acute barbiturate poisoning are drowsiness and mental confusion progressing to coma; but mild excitement, ataxia and slurring speech may occur and lead to a suspicion of alcoholic intoxication. Chronic intoxication may give vertigo, ataxia, diplopia and a whole galaxy of physical signs, so that organic neurological syndromes are simulated, e.g. disseminated sclerosis or Parkinsonism. Mentally these cases often resemble chronic alcoholics. Isbell regards them from the point of view of the community as more dangerous than morphine addicts.

Phenobarbitone is specially liable to give severe and sometimes fatal skin eruptions. Two of my colleagues at Sheffield[4] have recently reported 4 such cases, 2 of which died. The eruption continues to spread when the drug is stopped and no specific changes are found at autopsy. The amount of phenobarbitone given may be quite small—as little as ½ grain 3 times a day.

Treatment of Acute Poisoning

The best treatment of acute barbiturate intoxication is still under discussion. The standard treatment consists in stomach wash-outs and the administration of picrotoxin or a similar analeptic. Penicillin is given as a prophylactic against hypostatic pneumonia. Recently claims have been made for a form of treatment similar to that used in anaesthetic overdosage, viz. intubation or suction, oxygen therapy and blood transfusion[5]. It is said that the awakening effect of the analeptics is not sustained in cases where doses well above anaesthetic doses have been taken and that they lead to depression after initial stimulation.

References

1. Jetter, W. W. and McLean, R. (1943): *Poisoning by the Synergistic Effect of Phenobarbital and Ethyl Alcohol*, Arch. Path., **36**, 112.
2. Fisher, R. S., Walker, J. T. and Plummer, C. W. (1948): *Quantitative Estimation of Barbiturates in Blood by Ultra-violet Spectrophotometry*, Amer. J. Clin. Path., **18**, 462.
3. Paxson, N. F. (1932): *Obstetrical Anaesthesia and Analgesia with Sodium* **iso**-*Amylbarbiturate and Nitrous Oxide-Oxygen*, Anaesth. and Analg., **11**, 16.
4. Sneddon, I. B. and Leishman, A. W. D. (1952): *Severe and Fatal Phenobarbitone Eruptions*, Brit. Med. J., **1**, 1276.
5. Nilsson, E. (1951): *On the Treatment of Barbiturate Poisoning*, Acta Med. Scand., Supp., 253.

Estimation of Barbiturates

By the Use of Ultraviolet Spectrophotometry

J. T. Wright, D.M. (Oxon.), M.R.C.P., (Lond.)

Medical Unit, The London Hospital, London, England

In collaboration with Mr. R. G. S. Johns, Principal Biochemist to the Clinical Laboratories at the London Hospital, I have examined a number of cases of barbiturate poisoning over the past 18 months, using an ultraviolet spectrophotometric technique for measuring blood, tissue and body fluid barbiturate levels.[13]

The principles of this technique were published by Stuckey[8] and by Elvidge[3] as long ago as 1940. Stuckey[9] used the method as a check on standard methods of barbiturate assay in forensic cases, but apart from the use of a similar technique in the assay of the thiobarbiturates, the method does not appear to have been applied to clinical problems involving ordinary barbiturate derivatives until 1948.[5, 6, 11] Born,[2] of University College Hospital, independently discovered a similar method in the following year and Lous of Copenhagen[7] modified existing methods.

Despite Born's contribution it appears that the method has not been widely used in this country until very recently, probably owing to the lack of availability of suitable spectrophotometers. The principle of all methods depends on the fact that barbiturates in alkaline solution have a characteristic ultraviolet absorption spectrum. Most methods of assay also make use of the fact that in acid solution barbiturates (with certain exceptions) absorb negligible quantities of ultraviolet radiation.

Fig. 1A compares the absorption spectra of phenobarbitone and barbitone (Fig. 1B) in alkaline and acid solution. In addition these figures show that barbiturates recovered from the urine of patients poisoned by phenobarbitone and barbitone compare very favourably as regards their ultraviolet spectra with equal weights of pure commercial products.

Fig. 2 shows that though extracts of normal blood absorb a certain amount of ultraviolet radiation, this absorption is for practical purposes independent of pH at the wave length which is critical for barbiturates (239—240 mμ at pH 10). It is thus possible to make use of the difference in absorption of ultraviolet light in alkaline and acid solution at this wave length for the assay of these substances in blood.

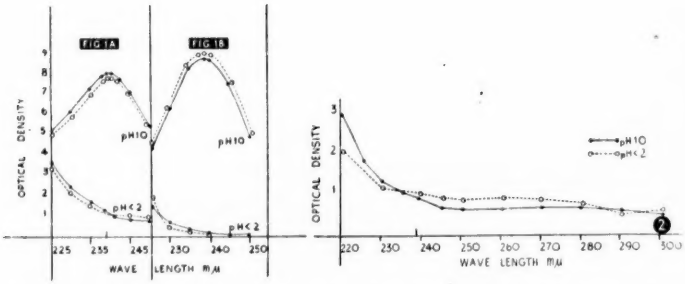


Fig. 1. A and B ultraviolet spectra in alkaline (pH 10) and acid solution (pH less than 2) phenobarbitone and barbitone respectively.
Continuous lines indicate spectra of samples obtained from urine of poisoning cases. Broken lines represent spectra of commercial products.
Fig. 2. Composite absorption spectra in alkaline and acid solution of extracts of 5 c.c. samples of blood from 10 normal people.

Fig. 3 shows the absorption spectra of liver, blood and kidney extracts from a fatal case of phenobarbitone poisoning. The heights of the peaks do not indicate the relative concentration of phenobarbitone in the various tissues since the amounts of tissue extracted have not been adjusted in this case.

Fig. 4 indicates how the blood barbiturate level may be followed in a case of poisoning. The patient, an epileptic aged 49, took 42 gr. of phenobarbitone 2 hours before admission. He was in moderately deep coma until the evening of the second day, when he was rousable with difficulty. Thereafter he exhibited slowly decreasing drowsiness, dysarthria, diplopia and nystagmus until the 6th day when he became free of symptoms. On

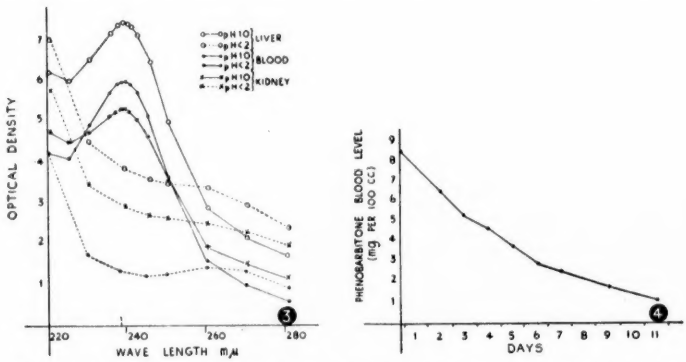


Fig. 3. Absorption spectra in alkaline and acid solution of extracts of blood, liver and kidney from a fatal case of phenobarbitone poisoning (Dr. Camps' case).
Fig. 4. Chart showing daily blood phenobarbitone levels in a male epileptic aged 49 who took 42 gr. of phenobarbitone 2 hours before admission.

the morning of the 7th day he had three major epileptic fits and on the morning of the 11th day he became violent and was transferred to an observation ward.

There are several limitations to this method. Firstly, in my opinion, it does not reveal the type of barbiturate concerned, though some authors claim that there are detectable differences in the slope of the absorption curves, even though the peaks occur at the same wave length. Very recently[6] a method has been described in which it is claimed that minute quantities of barbiturate can be detected and the actual barbiturate derivative can also be determined. We have not yet investigated this method. A further limitation of our method is that barbiturate can only be detected for certain when it is present in a concentration appreciably in excess of interfering chromogens. With 5 c.c. blood samples we get reliable results down to a concentration of barbiturate of 2 mg. per 100 c.c., though we can make a reasonable estimate down to 1 mg. per 100 c.c. This is unfortunate because subjects poisoned by short-acting barbiturates may be dangerously depressed and yet have blood levels of the order of 1–2 mg. per 100 c.c. One young athletic man on whom Dr. Camps recently performed a post-mortem examination had a blood pentobarbital level of only 3.2 mg. per 100 c.c., though alcohol was probably a contributory factor in this case. Fatal cases, usually alcoholics, with lower blood levels have been reported in the literature.[4, 5]

Fig. 5 shows the spectrum of a urinary extract illustrating the effect of

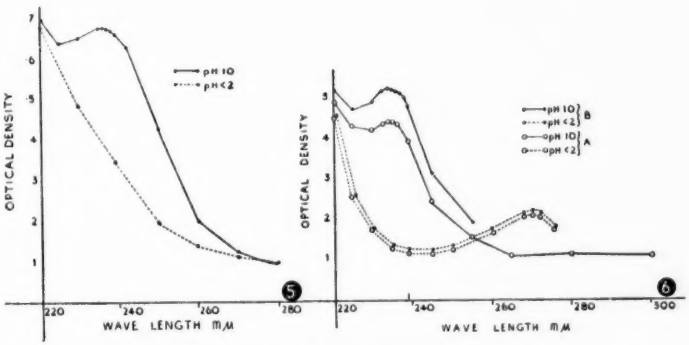


Fig. 5. The absorption spectra of a diluted extract of the patient's (Fig. 4) urine passed on the third day. Note alkaline absorption peak at 236 mμ.

Fig. 6. A. Absorption spectra of a normal blood in which commercial heparin had been used as an anti-coagulant.
B. The same with addition of 20 micrograms of barbital.

interfering chromogens in mild degree. The alkaline absorption peak occurs at 236 mμ rather than 239 mμ. The importance of a guarded opinion when there is an abnormal peak is shown in Fig. 6, Curve A being the ultraviolet spectrum of an extract of normal blood in which a small amount of com-

mercial heparin had been used as an anti-coagulant. The whole of the absorption spectrum in alkaline solution is due not to barbiturate, but to the minute traces of phenolic preservative present in the heparin. Curve B is an extract of the same blood to which a small amount of barbiturate has been added. It will be seen that the maximum alkaline peak is still at 234 mμ. The presence of this phenolic contaminant is suggested, however, by the smaller peak which appears at about 270 mμ in acid solution. This illustrates the importance of examination of the whole spectrum from 220 mμ to 300 mμ. If this is done, commonly occurring interfering substances such as sulphonamides and salicylates will be recognized.

The possible pitfalls of the spectroscopic method of identification of barbiturates are as yet probably insufficiently recognized for the method to be used alone in important forensic cases. Coupled with some qualitative method of identification such as Turfitts micro-crystalline method it should, however, prove very useful; and where very small amounts of barbiturates are concerned it is likely to be of considerable value when used in conjunction with paper chromatography.[1, 12, 14]

References

1. Algeri, E. J., Walker, J. T. (1952): Amer. J. Clin. Path., **22**, 37.
2. Born, G. V. R. (1949): Biochem. J., **44**, 501.
3. Elvidge, L. F. (1940): Quart. J. Pharm. Pharmacol., **13**, 219.
4. Fisher R. S., Walker, J. T. and Plummer, C. W. (1948): Amer. J. Clin. Path., **18**, 462.
5. Goldbaum, L. R. (1948): J. Pharmacol. Exp. Therap., **94**, 68.
6. Goldbaum, L. R. (1952): Analyt. Chem., **24**, 1604.
7. Lous, P. (1950): Acta. Pharmacol. Toxicol., **6**, 227.
8. Stuckey, R. E. (1940): Quart. J. Pharm. Pharmacol., **13**, 312; **14**, 217; **15**, **370**, 377
9. Stuckey, R. E. (1952): Personal communication.
10. Turfitt, G. E. (1948): Quart. J. Pharm. Pharmacol., **21**, 21.
11. Walker, J. T., Fisher, R. S. and McHugh, J. J. (1948): Amer. J. Clin. Path., **18**, 451.
12. Wickstrom, A. and Salvesen, B. (1951): J. Pharm. Pharmacol., **4**, 98.
13. Wright, J. T. and Johns, R. G. S. (1953): In the press.
14. Wright, J. T. (1953): In the press.

MEDICO-LEGAL NOTES AND NEWS

American Academy of Forensic Sciences

The Sixth Annual Meeting of the American Academy of Forensic Sciences will be held at the Drake Hotel, Chicago, from 25 to 27 February 1954.

South African Medico-Legal Society

This Society was constituted originally as the Medico-Legal Society (Johannesburg). In August 1952 Mr. Justice Greenberg delivered the Inaugural Address. Thereafter the following meetings were held:

"Race in Science and Law" (on 10 November, 1952);

"Medico-Legal Mythology: Some Popular Fallacies" (on 26 February 1953);

"Negligence in Medical Practice" (on 10 June 1953);

"The Medico-Legal Aspects of Head Injuries" (on 25 August 1953); and

"The Modern Approach to Legal Responsibility, the Psychopath, and the M'Naghten Rules" (on 4 September 1953).

Journal of Forensic Medicine: The Society concluded an arrangement with the publishers of this *Journal* whereby members of the Society receive copies of this *Journal* as part of their membership privileges.

A meeting of the Society was held in January 1954, when Dr. Yellowlees spoke on *Guilty but Insane.*

Those interested in joining the Society should communicate with the Honorary Secretaries at P.O. Box 6434, Johannesburg, South Africa. Special facilities exist for overseas members.

British Association of Forensic Pathologists

The Summer Meeting of this Association was held on 27 and 28 June 1953 at the Retsmedicinsk Institut, Copenhagen.

The Seventh Meeting of the Association was held at the London Hospital Medical College on 28 November 1953. The agenda included a symposium on *Anoxia and the Interpretation of the Post-Mortem Appearances.*

REVIEWS OF BOOKS

INSANITY-PRODUCING DRUGS AND THE LAW

The Hallucinogenic Drugs (The Insanity-Producing Drugs: Indian Hemp and Datura)—A Neglected Aspect of Forensic Medicine (A Loophole in the Law). By Donald McI. Johnson, M.A., M.B., B.Ch., Barrister-At-Law. (Pp. 45, 55.) London: Christopher Johnson Publishers Ltd. 1953.

Contents: Part I: Introduction. The Nature of the Hallucinogenic Drugs. Forensic Medicine: A Neglected Aspect. Criminal Drugging by the Hallucinogenic Drugs in the East. Can Criminal Drugging by Insanity-Producing Drugs Occur in Western Society and what would be its Results?

Part II: Personal Observations. A Psychotic Episode. An Epidemic of Intoxication Mania in the Nineteen-Fifties. A Case of Mania through Marihuana Intoxication in the United States.

Part III: The Case of Mrs. Cynthia Elsie White. Summary. Appendices. Bibliography. Glossary.

Dr. Donald McI. Johnson, Medical Practitioner and Barrister-at-Law, 'can claim to have achieved the dubious distinction of being probably the first medical man to have been certified as of an unsound mind by a fellow medical man under the new Section 14 of the Lunacy Act, as amended by the Ninth Schedule of the National Health Act of 1946, (p. 27).

In the course of a busy practice, and while in good health, the author 'went into a state of mental excitement, violent physical agitation, hallucinations and incoherence, while euphoria and laughter alternated with intense feelings of anxiety' (p. 28). The author's wife, of normal mental and physical health, 'simultaneously manifested identical symptoms' (p. 28), but was not certified, being left in the care of relatives, and 'without further medical observation or treatment'. Both the author and his wife made complete recoveries within a reasonably short time.

The author takes the reasonable view, for which he marshalls adequate and convincing evidence, that he was the unwitting victim of an acute poisoning episode, probably by one of the aptly-named hallucinogenic drugs (Datura or Indian hemp).

He points out that there is a considerable hiatus in our organized, co-ordinated knowledge in this field. This situation his present contribution should do much to retrieve.

Dr. Johnson's experiences stress the almost sinister bureaucratic powers which can be wielded in terms of the National Health Act of 1946, which clearly requires serious amendment.

An important Addendum draws attention to the recently-disclosed effects of lysergic acid diethylamide (LSD) and its power to produce the clinical picture of schizophrenia.

Dr. Johnson also makes out a good case for the view that the notorious epidemic of bread poisoning in France (Port Saint Esprit) was due to LSD, an alkaloid related to ergot.

This is an important monograph which emphasizes the modern armamentarium available to the poisoner who can send 'his victim into schizophrenia rather than mania, if he so wishes'.

BLOOD GROUPS MADE EASY

The Human Blood Groups Utilized in Disputed Paternity Cases and Criminal Proceedings. By P. H. Andresen, Chief, Municipal Blood Bank, Bispebjerg Hospital, Formerly Chief, Serological Department, University Institute of Legal Medicine, Copenhagen, Denmark. (Pp. 114-viii.) Springfield, Illinois: Charles C. Thomas, 1952.

Contents: 1. Introduction. 2. General Description of the Blood Group Properties. 3. The Heredity of the Blood Groups in Brief Outline. 4. Medico-Legal Utilization of the Blood Groups in Disputed Paternity Cases. 5. The Practical Application of Blood Group Determination in Disputed Paternity Cases in Denmark. 6. Other Medico-Legal Aspects of the Blood Groups. 7. The Reliability of Blood Group Determinations Considering Each Group Separately. Index of Special Terms.

Dr. P. H. Andresen is already well known for his contributions to medico-legal matters. His present monograph covers his own very special field of interest, as he is in charge of the Municipal Blood Bank in Copenhagen and was formerly in charge of the Serological Department of the University Institute of Legal Medicine in the same city.

Dr. Andresen has written for laymen as well as legal and medical practitioners, but his monograph makes it quite clear that the day of the dilletante in the blood group field is over. Indeed, as a result of the impetus given to research by the discovery of the Rh factor, blood grouping has developed into what is virtually a new speciality.

The careful statement of the limitations of blood group studies for forensic purposes makes this a valuable reference source, even for medical practitioners, who can approach the present account with some relief at the prospect of understanding the intricate (rather than difficult) situation which has developed.

Dr. Andresen's account also indicates in passing the important medico-legal implications which comparative racial studies have disclosed. In South Africa, e.g. the chances of detecting a Bantu blood are as high as 80:1 in certain circumstances (Dr. M. Shapiro, this *Journal*, 1953,**1**,5); but in the same racial group, errors may occur whereby subgroups of group A may be mistaken for group O. In the South

African Bantu, A_2 and other subgroups of A account for 40% of Group A, unlike Andresen's European figure of 25%. This is of considerable practical importance.

The potency of the saliva as a source of grouping of forensic interest is also well illustrated in the following (p. 93):

'. . . the blood group properties are extremely marked in saliva. Consequently, the tests of these receptors have acquired medico-legal significance. Very important are the determinations on the dried saliva on cigarette stubs. In Denmark the method has been used mainly in the examination of anonymous letters, since it is often possible to decide the group from the saliva on the gummed edges of the envelope and postage stamps (1/16 part of a stamp may be sufficient). The Medico-Legal Institute once published a case in which a man was charged with having written a number of anonymous letters. The examination of 12 letters revealed that the stamps were moistened with saliva from a secretor of Group A. The person charged was, indeed, of Group A and he was, moreover, a secretor, so that he could easily have been the writer of the letters. Nevertheless, he went on refusing to admit the offence, and passed the charge on to an acquaintance of his. The latter proved to belong to Group O and could, therefore, be excluded. This result was of particular importance, since some of the letters contained data, assumed to be known only by these two men. The man was convicted, but the Court had not given particular weight to the laboratory results. Another example showing the importance of group determination is that of a murder case in which vomitus found on the carpet was found to belong to someone else than the deceased, who was a secretor of Group B, whereas the vomitus proved to be Group O or from a "non-secretor". The murderer was found to be Group A, but since he was a "non-secretor", the vomitus might have come from him, which he also confessed during the trial.'

Blood, sweat and tears, as well as other bodily secretions, yield remarkably valuable forensic data as far as the blood groups are concerned. Dr. Andresen's monograph is one of the few compact and readable accounts available to the non-specialist for instruction and guidance in this fascinating field.

THE CHRISTIE CASE

Medical and Scientific Investigations in the Christie Case. By Francis E. Camps, M.D. (Pp. 243—xxiii, with 47 Diagrams and Photographs and 6 Coloured Plates. 30s.) London: Medical Publications Limited, 1953.

Contents: 1. Investigation Before the Trial. 2. Examination of the Four Bodies. 3. The Laboratory Investigations, by L. C. Nickolls. 4. Examination of the Bones from the Garden, by Dr. Richard J. Harrison. 5. Examination of the Skeletons in Respect of Sex, Age and Stature, by Dr. Richard J. Harrison. 6. Examination of the Jaws and Teeth, by Professor A. E. W. Miles and R. W. Fearnhead. 7. Times of Death and Burial of the Two Skeletons. 8. Exhumation of Beryl Evans. 9. Carbon Monoxide Poisoning. 10. The Trial and the Scott Henderson Enquiry. Appendices.

Seldom has a murder investigation involved so many and such difficult medical and scientific problems as did the Christie case. Christie, it will be recalled, was hanged for the murder of his wife, but before and during his trial a most complex and intricate story was unfolded involving the bodies of three other women murdered in the same house, as well as the skeletons of two more females recovered from the garden.

Christie's case was also unique in that at one stage the alarming suggestion was made that one Evans (previously convicted of the murder of his young child Geraldine) had been hanged wrongly as Christie had been, it was alleged, responsible for the murder of both Mrs. Evans and Geraldine Evans.

Following a question in the House of Commons, the Home Secretary ordered an Inquiry (held by Mr. J. Scott-Henderson, Q.C.) to report whether 'there is any ground for thinking that there had been any miscarriage of justice in the conviction of Evans for the murder of Geraldine Evans' (page 194).

The findings of the Inquiry (published on 13 July 1953) were: 'There can be no doubt about the responsibility of Evans for the death of his wife and child. There is nothing to suggest that his conviction of these two murders was false other than the belated and inconsistent statements of Christie that he killed Beryl Evans but not Geraldine Evans' (pages 195-196).

A murderer with so many victims to his discredit (Christie's tally exceeded even that of Haigh's) obviously raises important and difficult psychiatric problems. It is hardly surprising to find that Crown and the Defence experts differed about the diagnosis. For the Defence, Dr. J. A. Hobson of the Department of Psychological Medicine, Middlesex Hospital, concluded: 'Christie's personality characteristics, his past medical and personal history, the psychiatric symptoms and signs he exhibits at the present time, are quite typical of what is seen in the mental illness to which we attach the label "hysteria". The medical use of the word "Hysteria" is rather different from its common lay usage. We do not mean by it that the patient suffers from temper tantrums. Hysterical symptoms are very common indeed and hysterical patients vary widely in the severity of their symptoms. Many apparently normal people at times show reactions of a hysterical type and at

the other end of the scale there are some hysterical patients who can only be dealt with adequately in a mental hospital. I regard Christie as one of the most severe hysterics that I have ever met with in my psychiatric practice. In my opinion these severer forms of hysteria should be regarded as "disease of the mind" of the McNaughten Rules. There seems to be no doubt that at the time Christie committed the acts with which he is charged he knew what he was doing but it is possible that at the time of the act he did not know that what he was doing was wrong. Christie forgets or falsely remembers with such a facility that it is even probable that at the time of committing the act he would conveniently forget that he was doing something that was contrary to the law' (page 181).

On the other hand, in rebutting evidence, Dr. J. C. Matheson, Principal Medical Officer of Brixton Prison and Dr. Desmond Curran, Consulting Psychiatrist to St. George's Hospital and the Royal Navy, differed quite strongly. Dr. Matheson felt that: 'Christie was sane and had not suffered from hysteria. His mental condition while in custody was largely due to his appreciation of the peril in which he was standing and his apparent indifference was a defence mechanism of his mind, which, as already stated, was in his experience very commonly found in persons accused of murder.

The events before, during and after all the alleged murders appeared to form a consequential sequence, one event leading up to the next, and Christie's conduct was . . . at all times purposive . . . at the times of of the alleged murders Christie knew the nature and quality of the acts and he knew they were wrong' (page 189).

Dr. Curran stated: 'I would regard Christie as the possessor of a highly abnormal character rather than as a victim of disease. Nor am I completely satisfied that the murders were necessarily sadistic. There is nothing, for example, to suggest that a sadistic motive entered in the murder of his wife. Christie himself repudiated any sadistic motive, emphasizing that to hurt anybody was the last thing he ever wished to do or had done. Christie also explained the previous charge of malicious wounding by telling me he was swinging the cricket bat of the prostitute's son idly in the room and it by chance "caught her head". Conclusion: I could find no evidence that Christie at the time of the murder of his wife was suffering from any defect of reason due to disease of the mind, nor that he is so suffering now. His behaviour both before and after the murder of his wife and of the three prostitutes forms a sequence that seems to me to show that he knew what he was doing and knew it was wrong. I do not think Christie is medically insane and he is certainly not mentally defective. On the contrary he is above average in intelligence. I regard Christie as being the posessor of a highly abnormal character rather than as the victim of mental illness or disease. Christie in my opinion is fit to plead and to stand his tiral. He fully realizes his position and indeed discussed with me the various possibilities the future might hold for him. He told me that on principle he had always been opposed to the death penalty' (pages 193-194).

These conflicting psychiatric opinions indicate the interesting and contentious debate presented by the problem of Christie.

Dr. Camps, however, has surveyed much more than the intricate psychology of a murderer. A vast amount of investigation involving the assembly of the skeletons from the remains found in the garden turned out in the end not to be directly relevant at the trial. From the medico-legal point of view, however, these reconstructions, as reported in this volume, form a most invaluable and up-to-date survey of many important problems of identification. No forensic expert should fail to study carefully the skilful account which has been prepared relating to the sorting out of the different skeletons and the determination of the probable sex, age and stature. Some aspects of this interesting work have been dealt with by contributors to this Journal (Vol. I, No. 1) and these references are included in Dr. Camps' review.

There is also an extremely important chapter illustrating once again the medico-legal importance of dental pathology.

It is not possible to touch on all the other important topics surveyed by Dr. Camps but it is desirable to mention the important observations made about the survival of sperms in dead bodies and the growth of moulds, the latter being of particular interest in view of Dr. Gormsen's contribution in this issue of the *Journal* on the production of alcohol in dead bodies *post mortem*. The inferences about coitus at or just before the death of the victim, based on the *quantity* of sperms found in the vagina after death, are, however, highly arguable.

There is an extremely important survey of the identification of pubic and vulval hairs; this chapter adds much of value to our studies on the identification of human hairs from different parts of the body.

The standard of production is extremely high and the multitude of significant illustrations enhances the value of what is likely to be a definitive account of the intricate forensic problems investigated in the Christie case.

CORRESPONDENCE

Air-Embolism Occurring during Utero-Tubular Insufflation

A Note on Two Fatal Cases

To the Editor: The danger of fatal air embolism occurring during tubular insufflation of the non-pregnant uterus is, on the whole, not fully appreciated. This may be due to the fact that the textbooks in general use do not warn their readers that the procedure may cause air embolism. It is a fallacy, common amongst some practitioners, that air embolism will only occur in the pregnant, but never in the non-pregnant uterus. Some medico-legal textbooks do not mention the possibility that air embolism may occur during tubular insufflation. Other medical textbooks mention it but do not emphasize the danger of air embolism. Titus[1], like Berkeley and Bonney[2], mentions the possibility of air embolism, but is more concerned with the danger of Lipiodol embolism occurring during the forceful injection of Lipiodol into the uterine cavity and the tubes. Neuweiler[3] recommends the use of carbon dioxide because, with the use of air, cases of fatal air embolism have been reported. He states that a pressure exceeding 200 mm. Hg: may cause air embolism.

At Pietersburg Hospital 2 fatal cases of air embolism occurred whilst the Rubin test, using ordinary air, was being performed.

Case 1: A Native female, who complained about sterility, was admitted to the Pietersburg Hospital. She gave her age as 30 years. Under a general anaesthetic a utero-tubular insufflation was performed. She collapsed and died during the manipulation. The air pressure was reported not to have exceeded 200 mm. Hg.

Autopsy.—The body was well preserved and there was no evidence of decomposition. Her age was apparently 40—45 years, not 30 years as stated. The autopsy was commenced by opening the skull. Whilst the skull was being sawn through it was noticed that air bubbles were escaping. More care was then used. The cap of the skull was removed and, when the dura was reflected, it was noticed that the cerebral veins were filled with air bubbles. There was hardly any blood in the veins. Air continued to escape from the veins injured by the saw. Unfortunately the cranial contents were removed before the thorax was opened, and the heart and the big vessels were not examined *in situ* but removed for the examination. The right side of the heart was filled with frothy blood. The non-pregnant uterus, normal in size, felt very hard and appeared fibrous. In the uterine cavity, near the fundus, a small superficial abrasion ¼ inch × ½ inch was noticed.

Death was accepted as due to extensive air embolism occurring during utero-tubular insufflation, air having entered the venous system through a slight abrasion caused during instrumentation.

Case 2: An Indian female aged 32 years was admitted to hospital for a tubular insufflation under a general anaesthetic. The patient's uterus was first curetted and then the Rubin test was performed. Whilst the insufflation was in progress she collapsed and died. The air pressure used during the insufflation was reported not to have exceeded 140 mm. Hg.

Autopsy.—The body of the deceased was examined 4 hours after death. She was a heavily-built Indian female, masculine in appearance and with masculine distribution of the pubic hair. Her apparent age was 30 years.

The mistakes made during the autopsy in the previous case were avoided. Care was taken with the opening of the skull. As in the first case, air started to escape from various places where the skull was sawn through. On removing the cap, the dura was left in position and when reflected revealed that the cerebral veins were filled with air bubbles. Air continued to escape from injured veins. Without removal of the cranial contents the neck was carefully dissected and the thorax opened. The large veins exposed were filled with large air bubbles. An incision was made into the pericardial sac and the sac was then filled with water. The ballooned heart floated. Whilst pressing the heart under the water the distended auricle was punctured and air rushed out. The uterus was non-pregnant and appeared to be fibrous in texture. The uterine cavity showed evidence of a recent curettage.

Death was accepted as due to extensive air-embolism occurring during utero-tubular insufflation following a curettage.

Summary and Conclusions. In both cases the uterus appeared fibrous in texture and the uterine mucosa was injured. The extent of the injury varied appreciably in these 2 cases. In spite of the variation in the degree of uterine mucosal injury, the quantity of embolic air found at autopsy was about the same. Extensive air embolism may occur even with a minute uterine mucosal injury. All that is actually required is for one single vein or sinus to be severed, its lumen to remain patent and the air pressure to exceed that of the venous pressure. It is safer to use an absorbent gas such as carbon dioxide.

One should remember the advice of Neuweiler[3] who states: 'Insufflation carries certain risks, and the test should be made only after careful consideration of the condition of each individual case'.

I wish to acknowledge the permission of the Secretary for Health and of the Superintendent, Pietersburg Hospital, for permission to submit this communication for publication.

References

1. Titus, P. (1945): *Management of Obstetric Difficulties.* St. Louis, Mosby, 3rd. ed.
2. Berkeley, C. and Bonney, V. (1942): *A Textbook of Gynaecological Surgery.* 4th ed., London: Cassel.
3. Neuweiler, W. (1952): *Handbook of Gynaecological Diagnosis.* London: Heinemann.

Pietersburg,
Transvaal,
South Africa.

H. N. v.d. G. Gertenbach,
M.B., Ch.B.